THE American JOURNAL OF Surgery

SEPTEMBER 1955

THURSTON SCOTT WELTON, M.D., EDITOR

Radical Neck Dissection

Bronchopulmonary Diseases

SIXTY-FOURTH YEAR OF PUBLICATION

SEPTEMBER 1955

THE American JOURNAL OF Surgery

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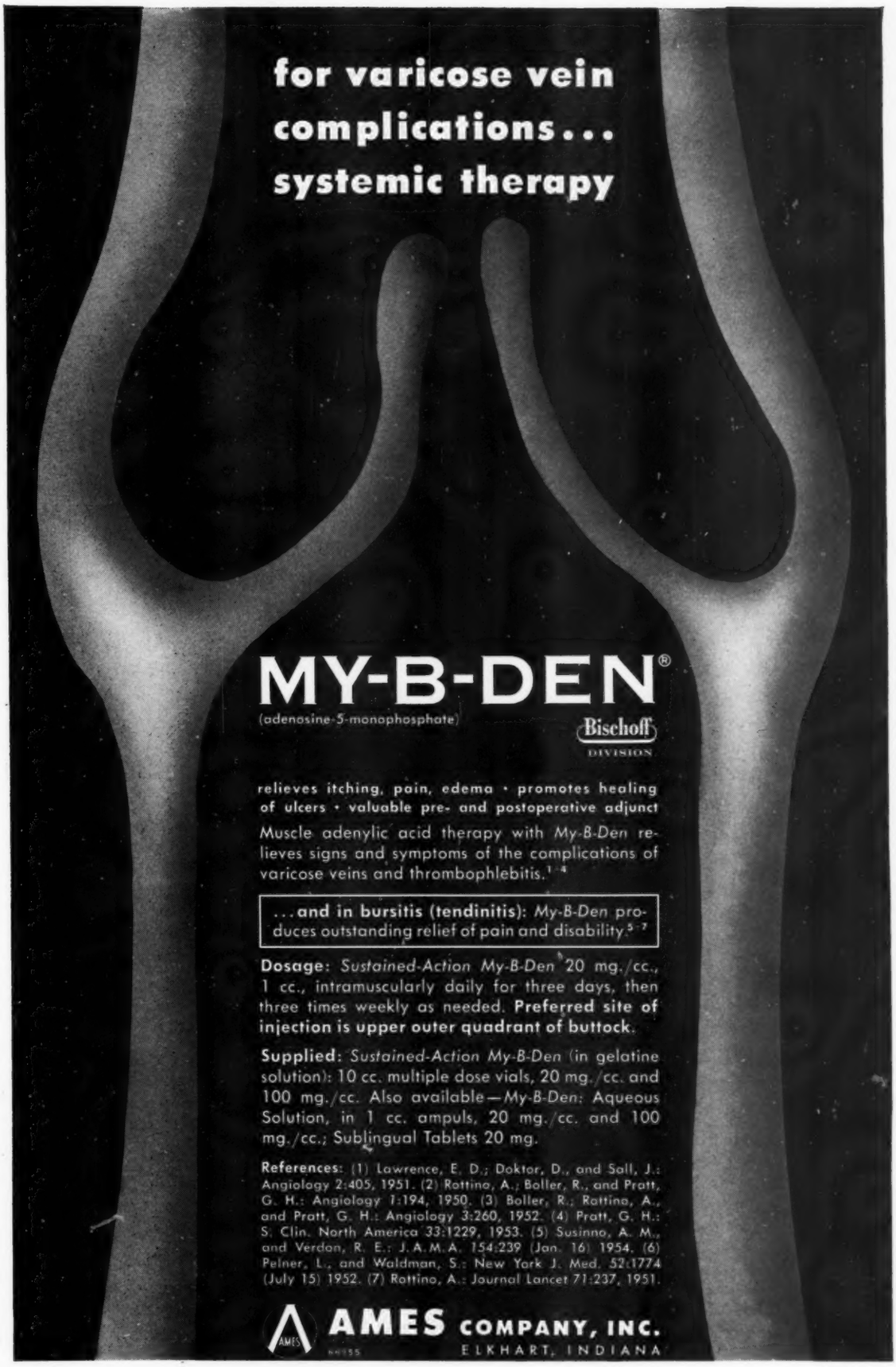
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SCIENTIFIC PAPERS

Spigelian Hernia 388

JOSEPH A. IGNATIUS

The subject of spigelian hernia has been discussed with reference to diagnosis, pertinent anatomy, possible etiology and treatment. Two case reports are included.

Portal Pressure and Medication by Percutaneous Splenic Route. An Experimental Study 392

FREDERIC W. TAYLOR AND DAVID C. GASTINEAU

In this study the spleens of experimental animals were transfixed beneath the skin to facilitate the study of portal pressures and cirrhosis. It was hoped that a fibrosing agent (silicon dioxide) could be indirectly introduced into the portal system by percutaneous injection and that this might result in cirrhosis with portal hypertension. Elevations formerly reported as portal hypertension are quite within the range found to be normal and physiologic.

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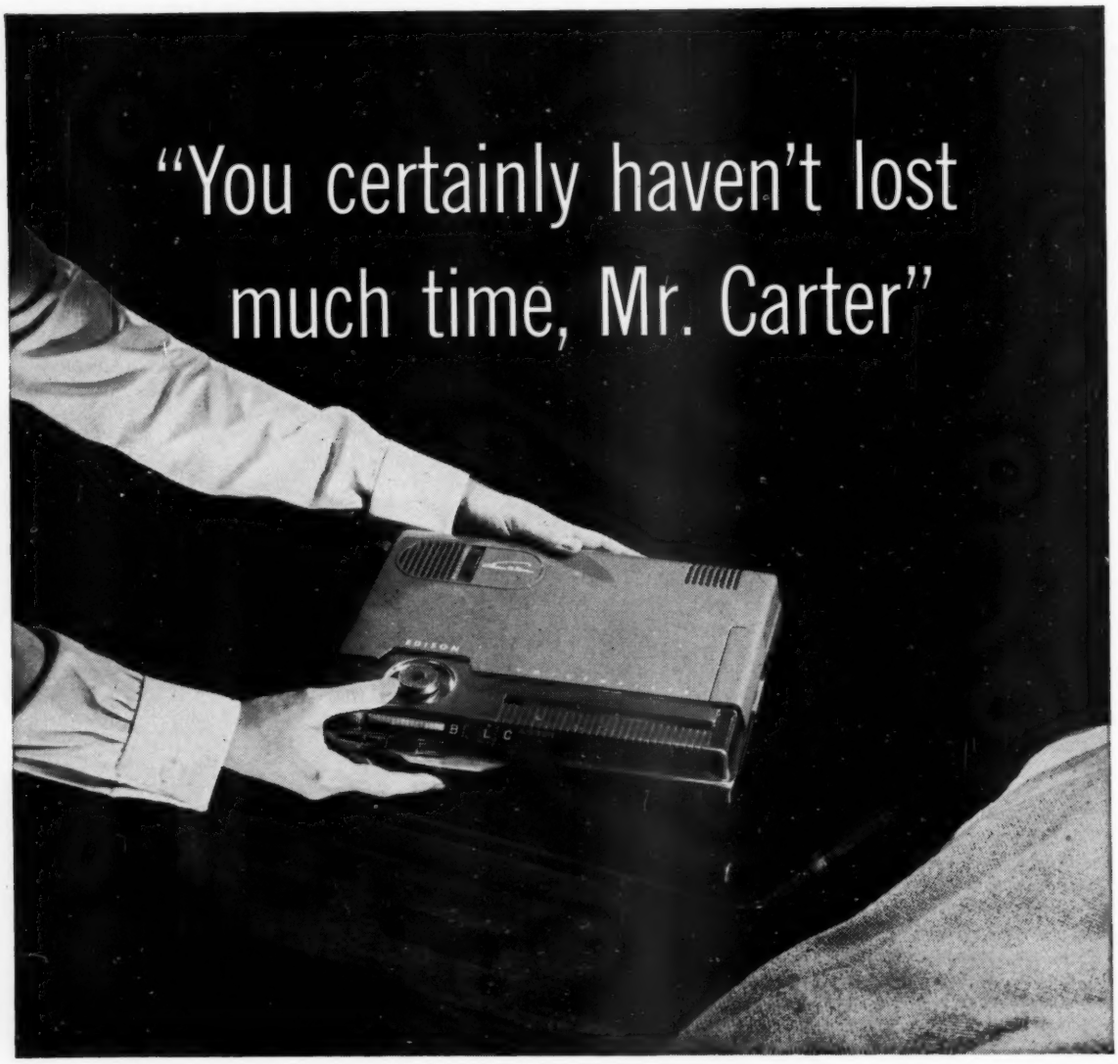

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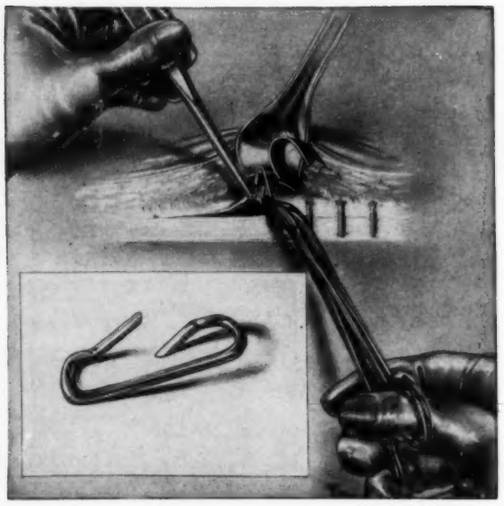

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By Paul I. Hoxworth, M.D.

Cincinnati General Hospital

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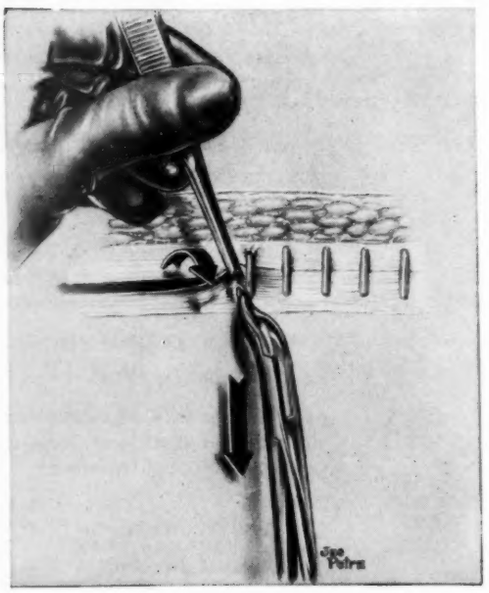

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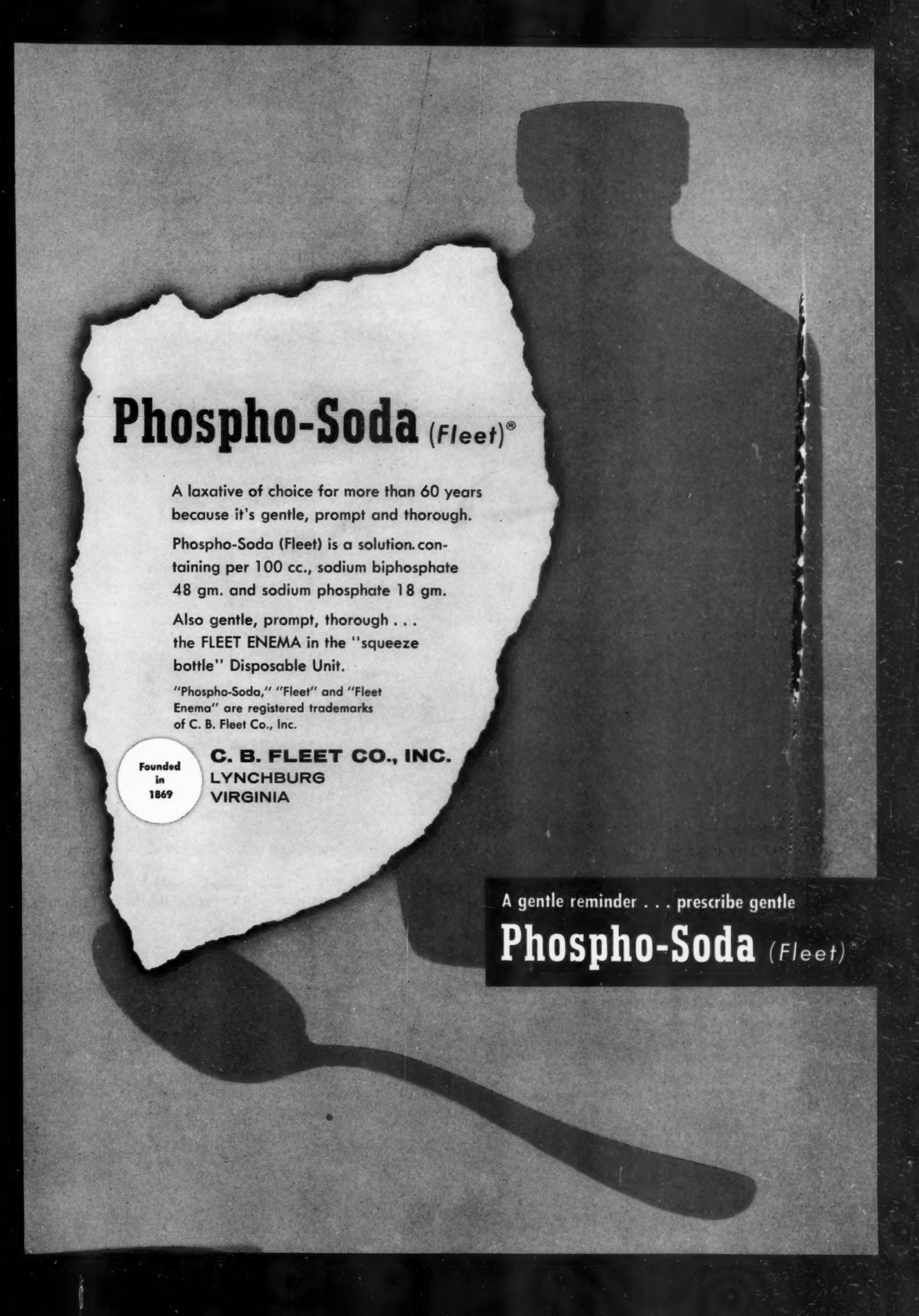
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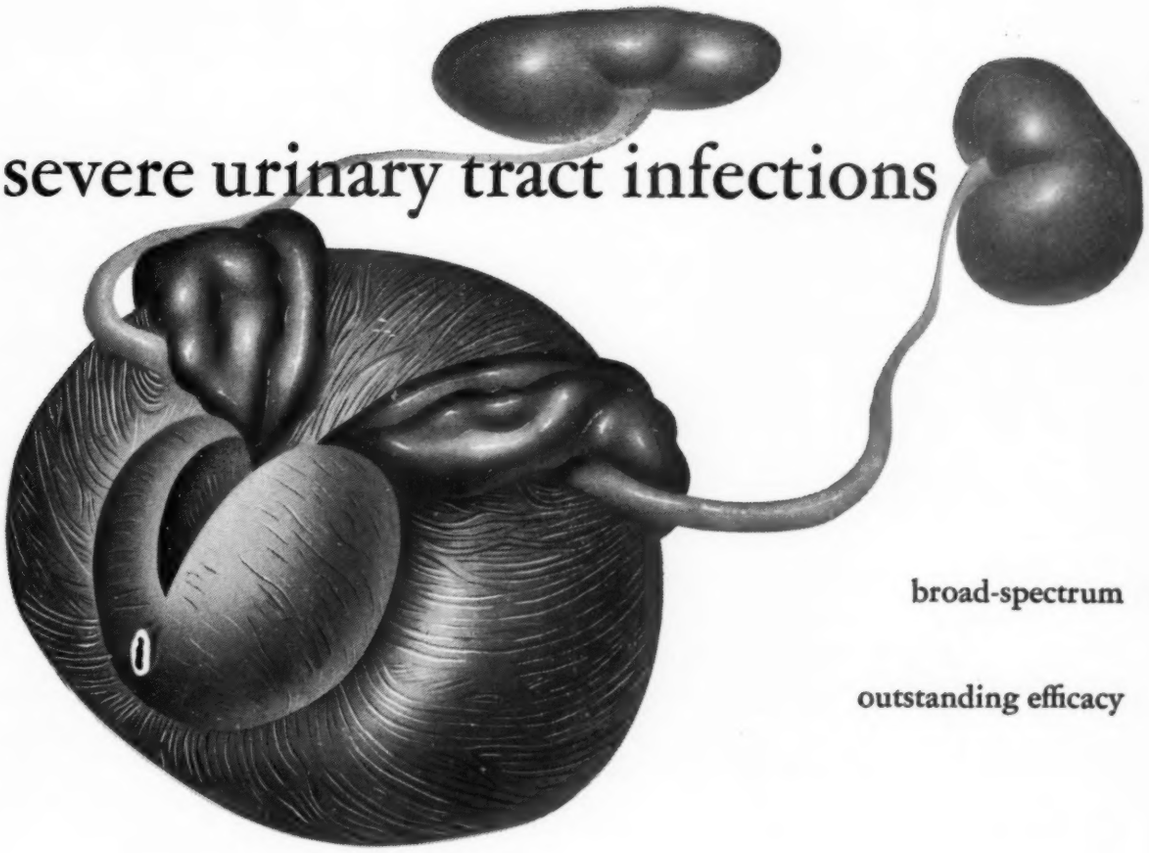

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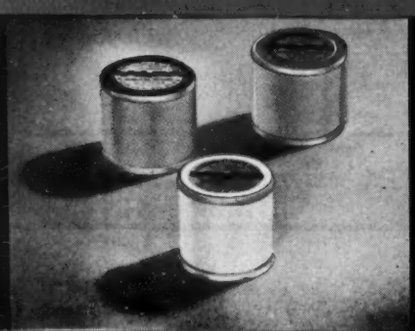

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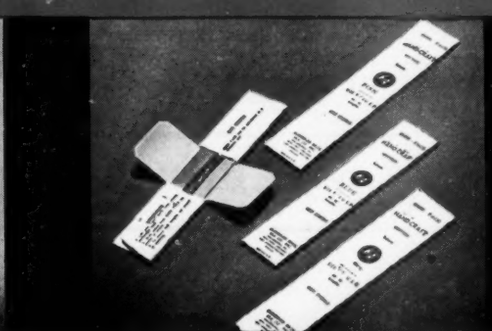

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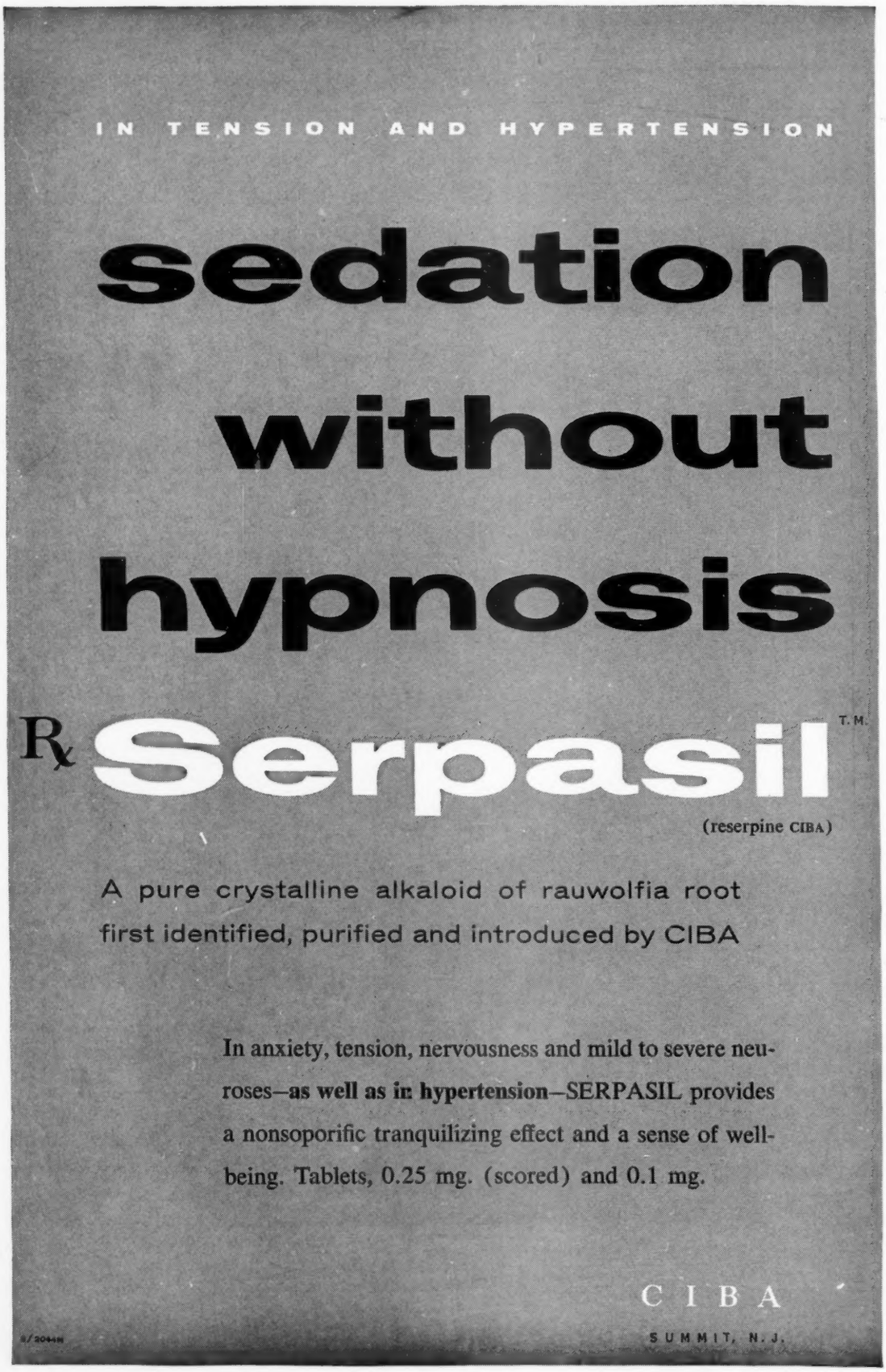
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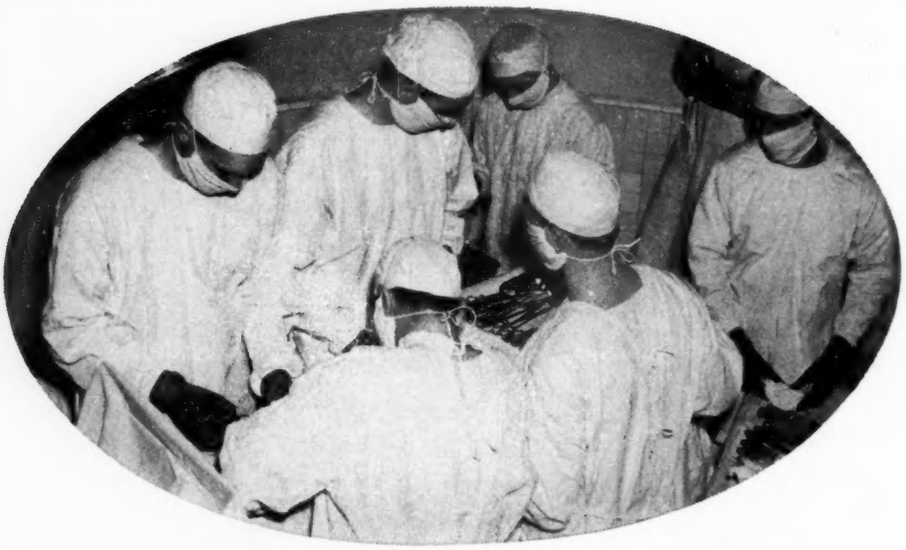

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1. Dumas, K. J.; Carlozzi, M., and Wright, W. A.: Antibiotic Med. *1*:296 (May) 1955. 2. Prigot, A.: Ann. New York Acad. Sc., in press. 3. Milberg, M. B., and Michael, M., Jr.: Ibid. 4. Pollack, H., and Halpern, S. L.: Therapeutic Nutrition, Prepared in Collaboration with the Committee on Therapeutic Nutrition, Food and Nutrition Board, National Research Council, Washington, D. C., 1952.

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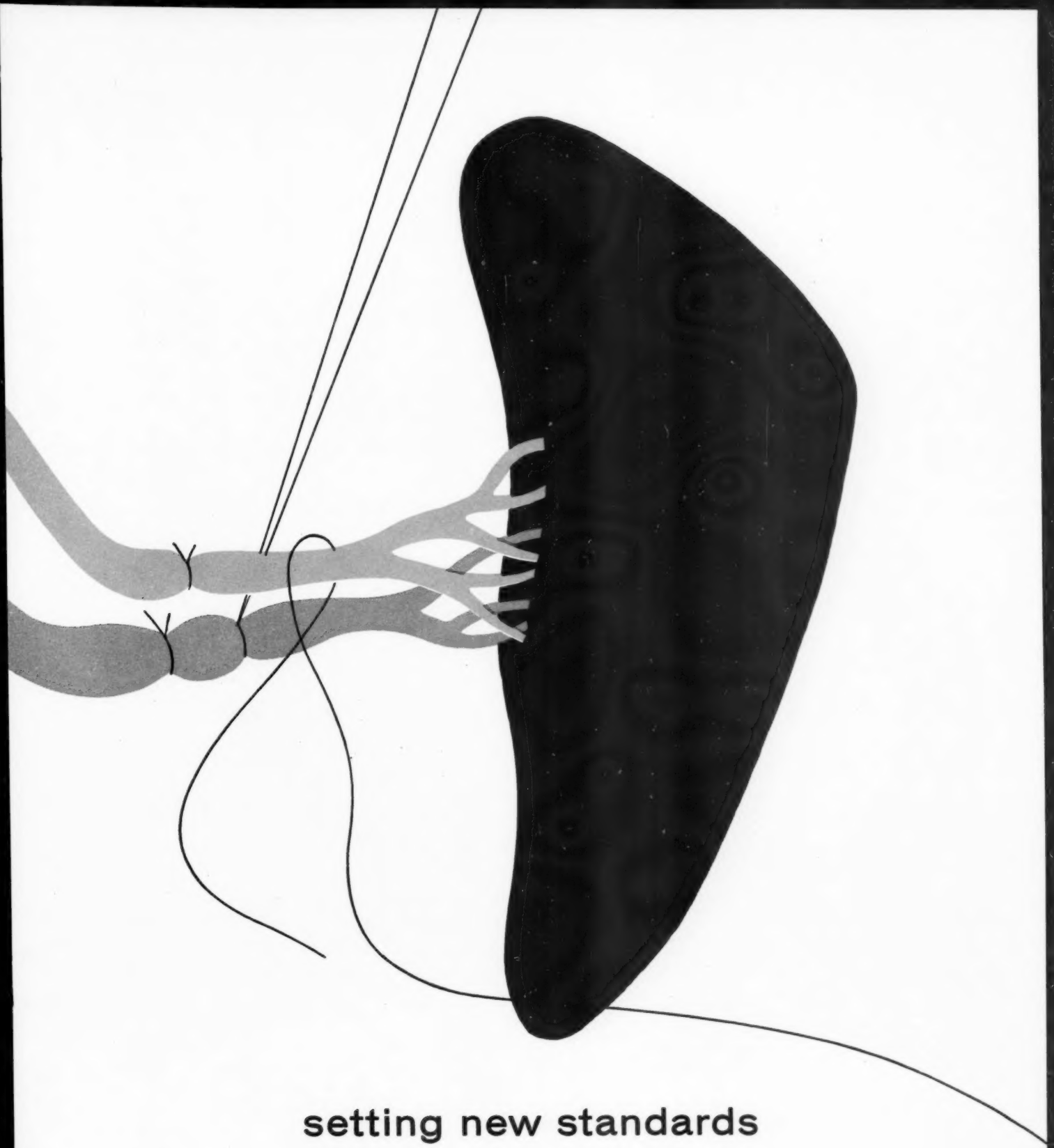
References: 1. Pharmacology and Therapeutics, ed. 2, Phila., Lea & Febiger, 1954, p. 452. 2. J.A.M.A. 153:1516, 1953.



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References: (1) Rosser, J. H., and McCall, R. A.: U.S. Armed Forces M. J. *5*:658, 1954. (2) Kantor, J. L., and Kasich, A. M.: Handbook of Digestive Diseases, ed. 2, St. Louis, The C. V. Mosby Company, 1949, p. 502. (3) Schwimmer, D.; Boyd, L. J., and Rubin, S. H.: Bull. New York M. Coll. *16*:102, 1953. (4) Best, R. R.; Rasmussen, J. A., and Wilson, C. E.: A.M.A. Arch. Surg. *67*:839, 1953.

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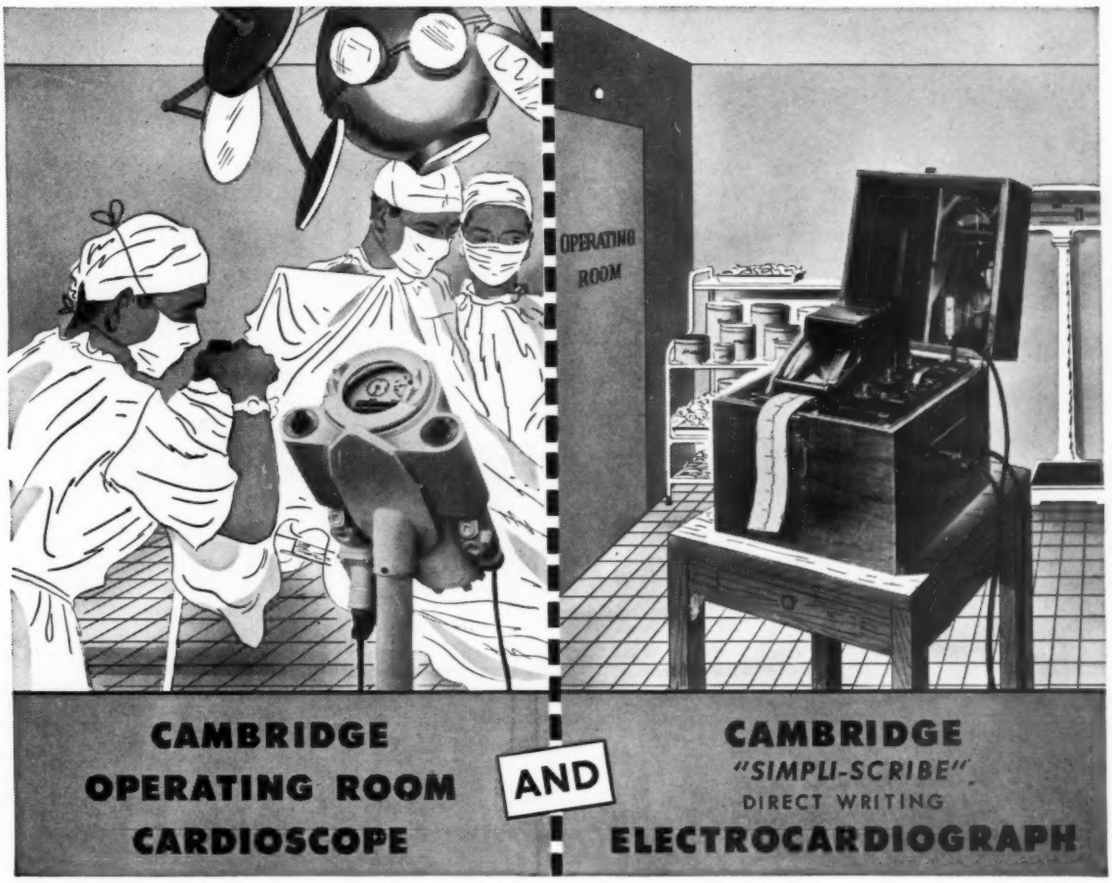

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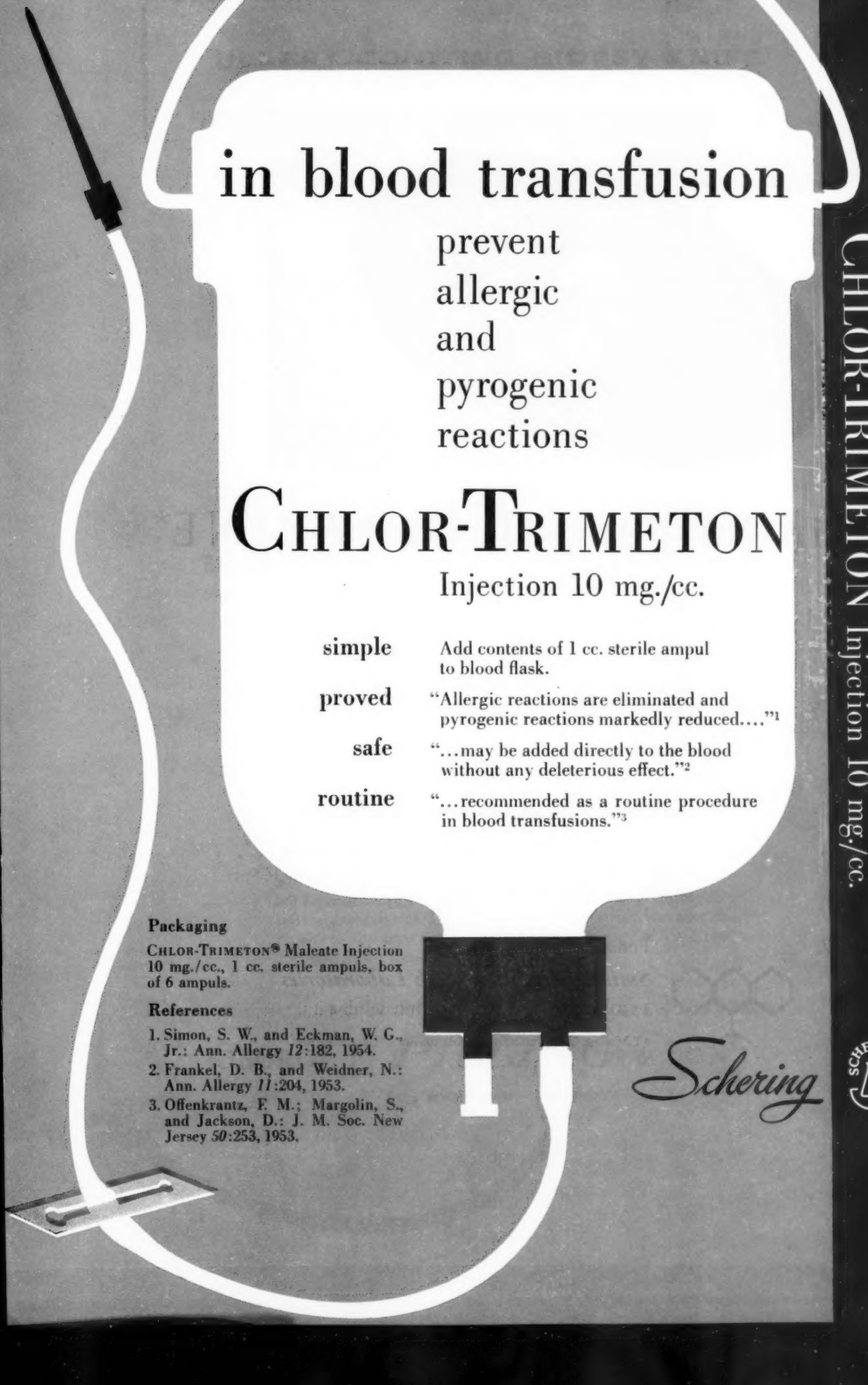

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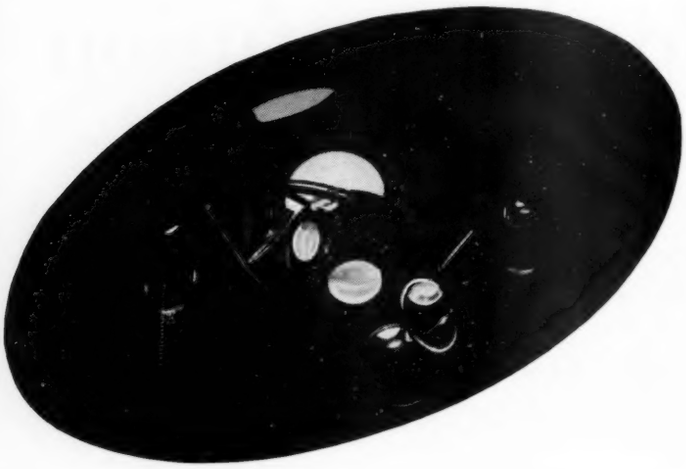

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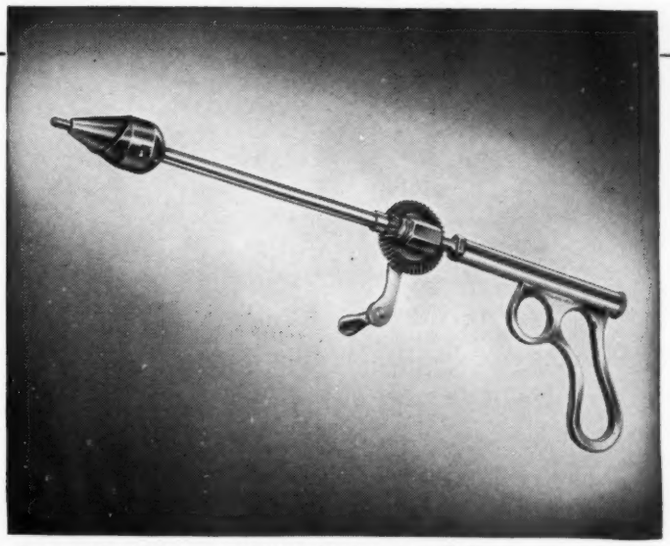

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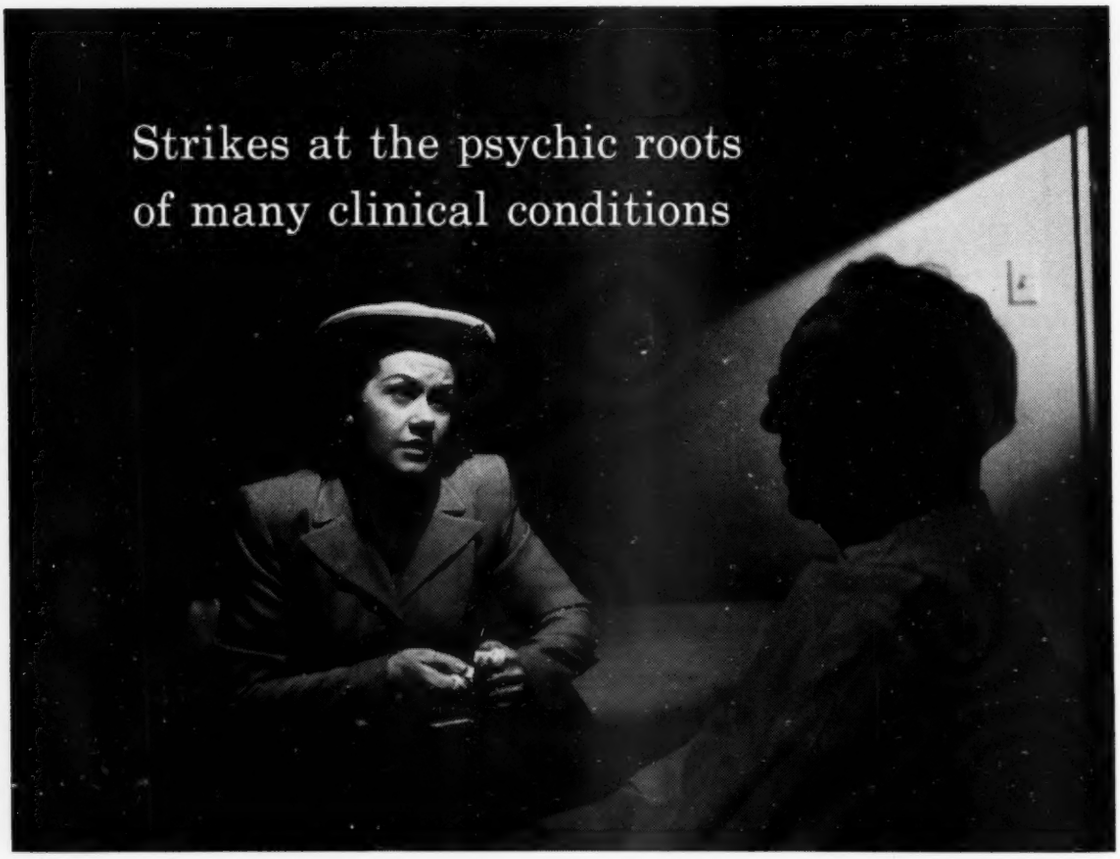

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VOL. 90 SEPTEMBER 1955 NUMBER THREE

Editorial

FOR THE WRITING PHYSICIAN

In the physician's daily routine he scans any number of journals that have come across his desk. He may find it necessary to do some writing of his own. In so doing he will, perhaps, begin to write an article for publication in a journal.

As soon as he is satisfied that the finished manuscript is the best he can do, he mails it to a journal, trusting it will be accepted. Perhaps the paper is subsequently returned with a polite note of rejection, in most instances offering no explanation of the journal's decision. Possibly the editorial office of the journal had on hand sufficient articles on the author's subject; it might have been textbook material saying nothing new; it could be that the author wrote on a subject long exhausted in the literature; or he might have presented a new method of treatment with only a few case reports as end results.

However, in many instances an article is rejected because it is poorly planned and written. Strange as it seems, it frequently happens that physicians who have bachelor and medical degrees often write like grammar grade students. Now and then, if the message is an important one, someone in the editorial office will rewrite the manuscript; but this is an exception to the rule.

If the young physician who desires to get his work into print is unsure of himself and is rusty on the rules of grammar and rhetoric, he would do well to read and study some of the books written by experts on the subject. We recommend "Words into Type," based on studies by Marjorie E. Skillin, Robert M. Gay and others, and "Medical Writing, the Technic and the Art," by Morris Fishbein, M.D.

Better still, he may communicate with the American Medical Writers' Association, which has recently announced a medical manuscript–editing service. This service is primarily for members, but non-members may submit manuscripts to them for criticism which is done by a faculty member of a well known school of journalism. Further details may be had by writing to the American Medical Writers' Association, Dr. Harold Swanberg, Secretary, 209–224 W. C. U. Building, Quincy, Illinois.

One of the most important goals of this Association is "to help maintain and to advance high standards of medical literature." We believe it would be profitable to become a member of this organization, thereby receiving their publications and obtaining much valuable information on medical literature.

Lucid and concise reporting is, of course, an excellent means of communicating ideas within the profession and to the laity. Surprisingly, it is easily accomplished.

T.S.W.

Spigelian Hernia

JOSEPH A. IGNATIUS, M.D., *San Francisco, California*

From the Department of Surgery, Stanford University School of Medicine, San Francisco, California.

SPIGELIAN hernia or spontaneous hernia through the semilunar line is of surgical interest largely because of its infrequent occurrence, the vague abdominal complaints which it may produce, and the occasional danger of strangulation. Two case reports are presented herein with a review of the pertinent anatomy, varied symptomatology and possible etiology.

The spigelian or semilunar line is a descriptive term denoting the transition of the transversus abdominis muscle into its aponeurosis which usually occurs slightly lateral to the lateral margin of the rectus abdominis. It apparently derives its name from the Flemish anatomist Adrian van der Spigelius (1578–1625). Its extent is from costal margin to pubic spine running along a gentle curve with its concavity away from the umbilicus. It may be visualized in the lean individual when the abdominal musculature is tensed. A spigelian hernia by definition, therefore, can occur at any point along this line. Koontz[3] reported seven instances of spigelian hernia, two of which occurred above the umbilicus. Mason and Ruge,[4] in their excellent review, cited a case in which the hernia was supraumbilical. However, most cases occur inferior to the umbilicus just lateral to the rectus at the junction of the semilunar line with the semicircular fold of Douglas, a fact that would seem to implicate this latter structure in the development of the abdominal wall defect. The fold of Douglas frequently forms the upper part of the aponeurotic defect and anatomically marks the caudal termination of the posterior rectus sheath. That the location of the semicircular fold is by no means constant has been demonstrated by McVay and Anson.[5] By dissections on fifty-six cadavers they found that the fold may occur at any level from umbilicus to pubic crest. A further finding was the presence of a secondary fold in twenty of the specimens studied.

Why the defect in the semilunar line occurs has not been satisfactorily answered. Koontz,[3] citing the work of Zimmerman et al.,[8] commented on how these authors demonstrated that the internal oblique and transversus abdominis muscles are frequently raised into muscular bands separated by weak aponeurotic tissues, providing sites for potential herniation. Other authorities suggest that the passage of nerves and branches of the inferior epigastric artery through the fascia and aponeurosis of the transversus abdominis provide routes for herniation, a theory applicable also to the formation of epigastric hernia. River,[7] in his comprehensive review of spigelian hernia, stated that there are numerous irregular, slit-like defects in the transversus aponeurosis, particularly at the point where the aponeurosis passes forward and fuses with that of the internal oblique. He cited the anatomic studies of other investigators supporting this, some of whom have found with frequency a lateral branch of the inferior epigastric artery passing through one of these defects. River believes that this artery is of little etiologic importance. Iparraguirre[2] cited two cases in which the hernia sac was accompanied with an artery which penetrated the transversus aponeurosis, presumably a branch of the inferior epigastric artery. Pampari[6] reported another case, confirmed by autopsy dissection, in which a conspicuous collateral branch of the inferior epigastric artery lay against the medial wall of the sac through the defect in the semilunar line. Anzilotti,[1] speculating on the pathogenesis of this hernia, believes that the presence of normal and abnormal vascular orifices in the transversus aponeurosis for the passage of the

inferior epigastric artery or its branches provides sites of anatomic weakness. It is apparent that there is no real agreement on this matter. However, immediate predisposing factors are, as River[7] states: congenital susceptibility, muscular atrophy, pregnancy, obesity, emaciation, chronic cough and severe muscular effort.

There is no characteristic symptomatology or significant difference in sex incidence. Complaints may be simply referable to a reducible abdominal mass unaccompanied with any discomfort. Other complaints are localized pain or discomfort which may be aggravated when standing or straining and improved on lying down. If strangulation occurs, a not uncommon complication, characteristic acute symptoms occur.

Examination of the patient will usually show a soft, reducible mass in the lower abdomen lateral to the border of the rectus, somewhat below the umbilicus. Following reduction of the mass an oval defect may occasionally be palpated in the abdominal wall through which the mass has herniated. This hernia is nearly always an interstitial one; that is, the presenting part of the sac lies beneath an intact external oblique aponeurosis, having penetrated the internal oblique and transversus. Thus there is a tendency for the hernia to be obscured or palpated with difficulty, particularly if the sac is small or if the patient is obese. In such instances the cause of the patient's complaints may not be readily apparent. A localized point of tenderness in the abdominal wall may in these cases be of diagnostic significance.

Operative correction should offer little difficulty in the uncomplicated case. The hernia is adequately exposed by a transverse or oblique incision over the presenting mass with division of the external oblique aponeurosis in the direction of its fibers. The hernia may then be visualized, frequently with a tuft of overlying properitoneal fat, penetrating the internal oblique aponeurosis. Opening the internal oblique aponeurosis allows for complete dissection of the sac. Its redundant portion is excised and the peritoneal defect sutured. The aponeurotic defect is then obliterated and the abdominal wall reconstructed utilizing the transversus aponeurosis, fold of Douglas and transversus fascia as a first line of defense, followed by reapproximation of the internal and external oblique aponeuroses.

CASE REPORTS

CASE 1. A fifty-seven year old woman entered Stanford University Hospital on January 30, 1951, with complaints of sensation of weight and of pulling in the right lower abdomen accompanied with a dull, throbbing pain and considerable audible peristalsis. These complaints had been present for the past year and were gradually becoming worse. She noted her symptoms were somewhat improved on lying down.

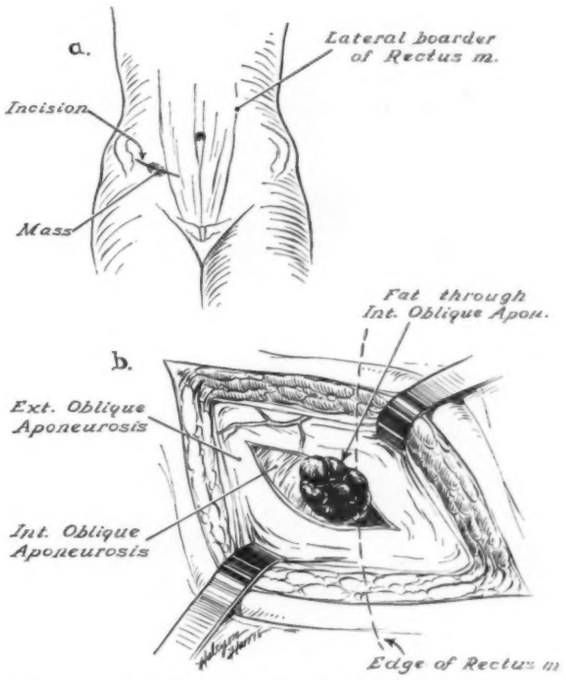

FIG. 1. (*a*) Location of hernia and incision. (*b*) Hernia sac presenting through divided external oblique aponeurosis.

Examination revealed a moderately obese patient with a tender, soft protruding mass in the right lower quadrant of the abdomen, located lateral to the margin of the rectus on a level with the mid-point of the inguinal ligament. The patient was operated upon on January 31st. (Fig. 1 to 3.) Through an oblique incision with division of the external oblique aponeurosis, a hernia sac covered with properitoneal fat was seen protruding through a defect in the internal oblique aponeurosis. Further dissection of the sac following opening of the internal oblique aponeurosis revealed that it came through a defect in the semilunar line just below the fold of Douglas. The sac measured 6 cm. long with a 4 cm. base. The

sac was excised and the peritoneum closed. The aponeurotic defect was then obliterated utilizing the fold of Douglas, transversus fascia and aponeurosis as a first line of defense followed by closure of the aponeuroses of the internal and external oblique muscles. The postoperative course was uneventful and the patient has remained free from recurrence to date.

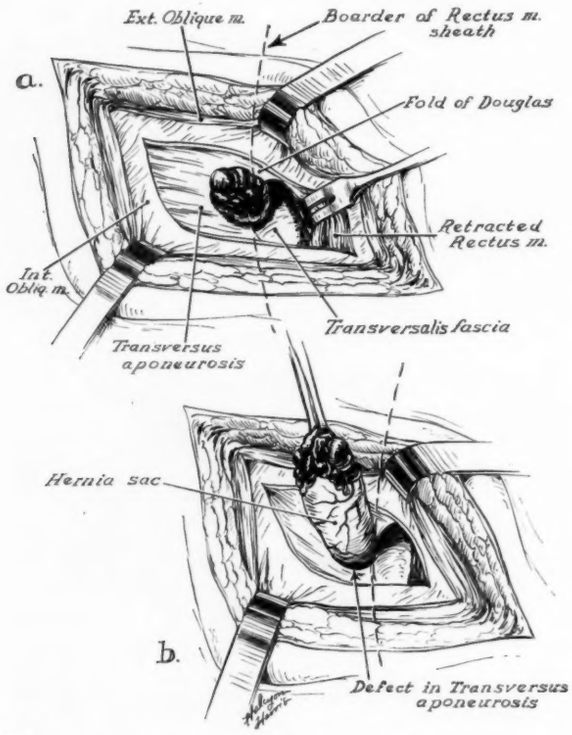

FIG. 2. (*a*) Internal oblique incised. (*b*) Completed dissection of the hernia sac showing pertinent anatomy.

CASE II. A seventy-two year old white woman entered San Francisco County Hospital on January 7, 1952, with a complaint of cramping abdominal pain and a painful mass in the right lower abdomen of twenty-four hours' duration. For the previous twenty-two years she had had an easily reducible mass in the right lower quadrant of the abdomen which had never been productive of acute symptoms until the evening before entry. At that time the mass appeared, became tender and could not be reduced. She felt nauseated but did not vomit. The mass finally reduced a few hours later but reappeared the following morning and was accompanied with cramping abdominal pain and tenderness. Her last bowel movement was twenty-four hours before entry; she had passed no gas per rectum for twelve hours.

Examination revealed a well preserved, elderly woman complaining of abdominal pain. There was a tender mass 8 cm. in diameter in the wall of the right lower abdomen lying above Poupart's ligament, just lateral to the border of the rectus. Peristalsis was hyperactive with occasional rushes. The white blood cell count was 13,300, with 79 per cent polymorphonuclear forms.

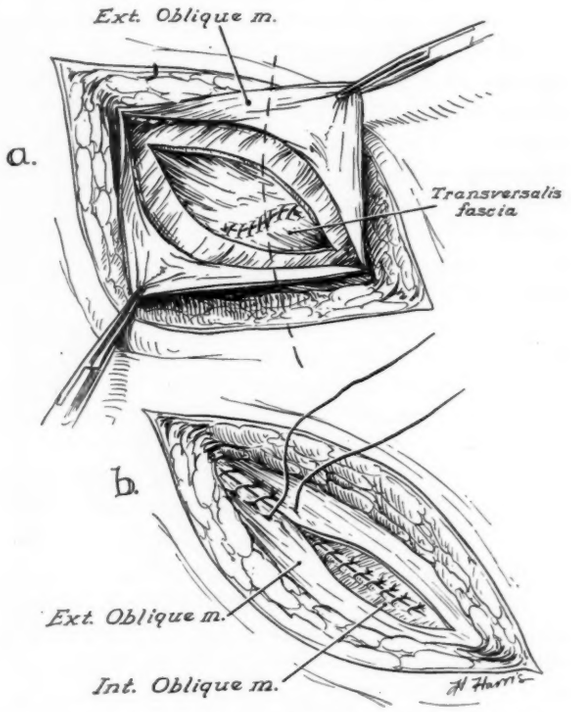

FIG. 3. (*a*) Sac has been excised and fascial defect closed. (*b*) Closure of the internal and external oblique aponeuroses.

A diagnosis of incarcerated spigelian hernia was made and the patient was operated upon shortly after entry. Through an oblique incision over the mass the aponeurosis of the external oblique was opened in the direction of its fibers. Lying between the aponeurosis and the internal oblique was a large edematous hernia sac which contained bowel. The sac was 17 cm. long and presented through a 5.5 cm. defect in the abdominal wall. This defect was located in the semilunar line at the level of the fold of Douglas. The sac was mobilized and opened and found to contain a moderate amount of bloody peritoneal fluid, several loops of dark purple small bowel and a portion of obviously

strangulated omentum. This portion of omentum was excised. After prolonged observation the appearance of the small bowel improved and resection was not necessary. Hence the sac was excised and the peritoneal defect closed. The fascia and aponeurosis of the internal oblique and transversalis muscles were closed over the defect. The external oblique aponeurosis was imbricated over this layer. The patient made an uneventful recovery.

SUMMARY

The subject of spigelian hernia has been discussed with reference to diagnosis, pertinent anatomy, possible etiology and treatment. Two case reports are included.

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Gumma of the breast is rarely seen today; however, when it occurs, biopsy is needed to exclude carcinoma. Usually an intact nodule is palpable, but if neglected it may break down and ulcerate. Some of the cases have been mistaken for a benign lesion or a tuberculous or malignant one, and unnecessary surgery has occasionally been performed, especially when biopsy was not possible or was forgotten. Adequate antiluetic treatment will handle gumma of the breast very nicely. (*Richard A. Leonardo, M.D.*)

Portal Pressure and Medication by Percutaneous Splenic Route

AN EXPERIMENTAL STUDY

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EXPERIMENTAL investigation of portal hypertension has been difficult because of the fact that animals had to be formally operated upon to obtain portal pressures or to administer drugs into the portal system. It would be quite advantageous to obtain portal pressures and give such injections without the necessity of laparotomy. The opening of the abdominal cavity of an experimental animal requires considerable time to merely uncover veins draining into the portal system. It also adds the unknown influence of anesthesia to a labile and uncertain portal pressure.

Some years ago one of us, in conjunction with Dr. Wemple Dodds, transfixed branches of the splenic vein of the dog to a position immediately beneath the skin. This was done so that repeated samples of splenic venous blood could be obtained easily. The method was only partially successful since the vein soon clotted with repeated puncture. Later it was suggested that possibly the spleen itself might be used in a similar manner for the present experiments. It was with this background that these studies were started. The spleens of mongrel dogs were transfixed subcutaneously beneath the skin in the left upper abdomen. In this position an exploring needle could easily and accurately be thrust into the splenic pulp without preliminary operation or anesthesia. Also, the danger of intra-abdominal hemorrhage following splenic puncture was minimized. Whatever hemorrhage might follow such a puncture would become a subcutaneous one. In the first few animals sodium morrhuate was injected beneath the skin covering the spleen so as to form a firmer attachment to its capsule. As the experiments proceeded, this appeared to be an unnecessary precaution.

Soon after this work was well under way a number of articles appeared in the literature indicating the ease and desirability of portal venography by both percutaneous and transabdominal routes.[1–5] The possibilities of this method as an experimental tool are interesting and it is thought that our experiences with it are worth recording.

METHODS

Under nembutal® anesthesia the spleens of six mongrel dogs were transfixed into a pocket beneath the skin of the left upper abdomen. The splenic pedicle was allowed to pass through an abdominal wall defect which did not cause constriction. At the time of operation portal pressures were obtained by inserting an 18 gauge needle into an omental or mesenteric vein. The needle was then attached to a three-way stopcock and a standpipe of saline. Portal pressure was taken by allowing the saline in the standpipe to fall to its natural level. The pressure thus obtained was corrected to the base pressure of the portal vein.[6]

Several weeks after splenic transplantation the dogs were again anesthetized and attempts were made to determine the splenic pulp pressure. This was done by means of the same type of gravity standpipe manometer, connected to an 18 gauge needle. The needle was thrust through the skin into the splenic pulp and the saline solution of the manometer was then allowed to seek its level. Corrections were again made to a base level of the portal vein. At times simultaneous portal pressures were taken with splenic pulp pressures for direct comparison. After these initial studies, pressures of the splenic pulp and the intrasplenic injections were

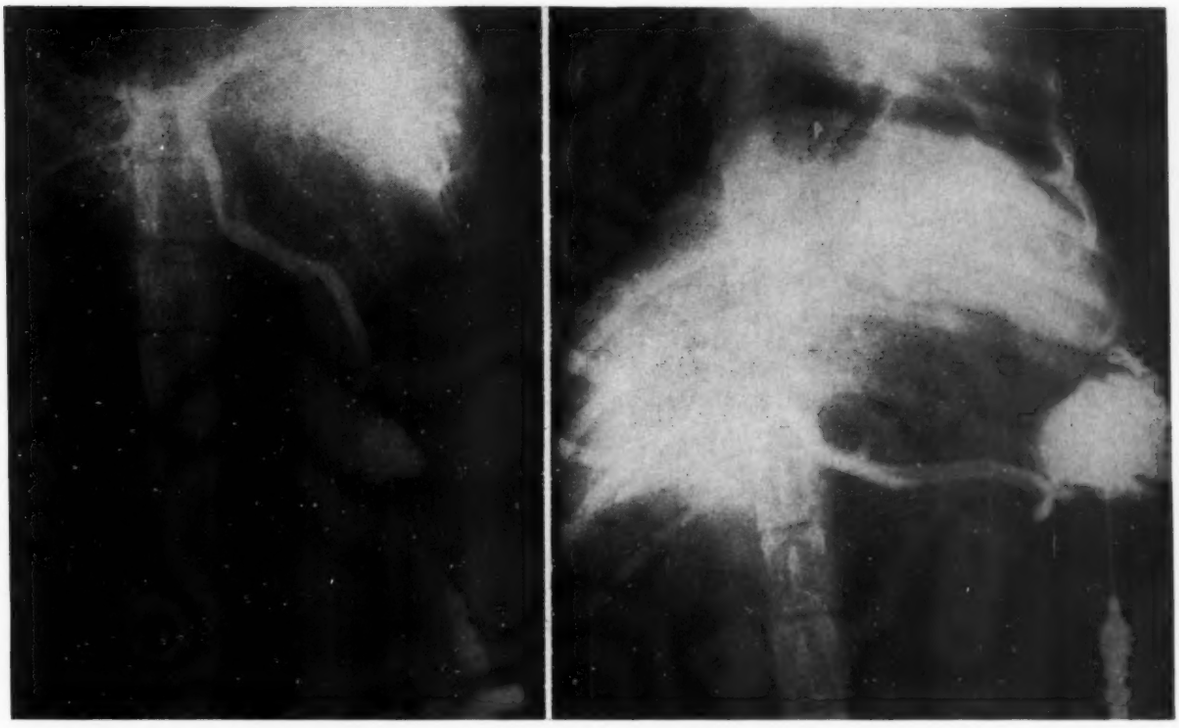

FIG. 1. Percutaneous injection of 70 per cent iodinized media into the subcutaneous spleen indicating the ease with which this material reaches the liver.

obtained without anesthesia and with the animals awake.

Attempts were made to visualize the portal system by injecting a concentrated iodized solution (75 per cent neo-iopax® or 70 per cent diodrast®) into the splenic pulp of the subcutaneous spleen. Ten cubic centimeters of the iodized solution were injected and an x-ray was taken just as the final amount of the solution left the syringe.

For some time we have been anxious to repeat the experiments reported by Rousselot and Thompson[7] in which silicon dioxide was injected into the portal system in order to produce a cirrhotic liver and portal hypertension. We therefore chose this percutaneous route of injecting the silicon. The material used was obtained through the courtesy of Dr. L. W. Spolyar of the Indiana State Board of Health. This quartz flour was 99.4 per cent SiO_2. Its particles varied in size from 1 to 50 μ. and by volume approximately one-half of these were under 3 μ. An amount of this quartz varying from 1½ to 2 gm. was placed in some 30 cc. of saline and this suspension was then injected into the splenic pulp. Since silicon dioxide is not radiopaque, it cannot be traced by x-ray. Its course was assumed to be the same as that taken by the radiopaque iodized solution.

These percutaneous injections were given at approximately six-week intervals and were carried on over a period of nine to eleven months. At the end of that time an average of 10½ gm. of silicon dioxide had been injected into the splenic pulp of each animal.

RESULTS

Portal Visualization. As noted by others,[1–5] the portal system was easily outlined by the injection of iodinized media into the splenic pulp. (Fig. 1.) Of nineteen such injections into the transplanted spleens, eighteen were successful in outlining the portal system although some were considerably less distinct than those illustrated. The single failure was the result of injecting the opaque material outside the capsule of the spleen. The best x-rays were those taken immediately after injection of the opaque media. However, this mass formed a small reservoir in the splenic pulp allowing the portal radicles to be visualized several minutes after injection.

Portal Medication. As previously mentioned, our prime interest in the foregoing injections

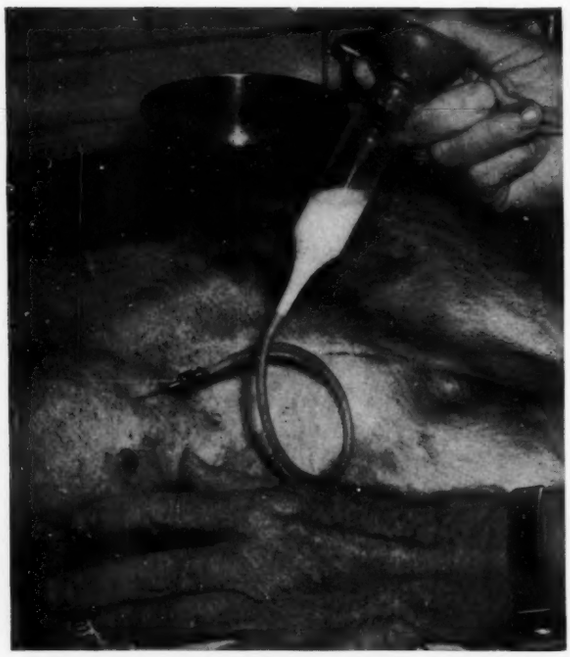

FIG. 2. Percutaneous injection of silicon dioxide into the spleen expecting it also to reach the portal radicles of the liver.

FIG. 3. Estimation of splenic pulp pressure. This roughly mirrors the portal pressure.

was to determine if other medications similarly injected might be expected to reach the liver by this route. Of special interest was the injection of silicon dioxide in an attempt to produce an experimental cirrhosis of the liver. This seemed to offer an attractive means of producing this lesion without the drudgery of repeated laparotomies. (Fig. 2.)

Since most of the quartz flour particles were less than 3 μ. in size, we had reason to believe that much of it would eventually reach the liver and cause the desired fibrosis. The immediate result of these percutaneous injections was a fibrosed and shrunken spleen in response to the foreign body irritant. In two there was actual infection (abscess). When splenic fibrosis occurred laparotomies were performed to see if liver scarring had taken place. The gross appearance of the livers of all six animals was quite smooth, soft and normal. Microscopic section of one was likewise normal. In three of the dogs there were enlarged lymph nodes near the hilum of the liver. Whether these were of any significance was not determined.

It is fully realized that cirrhosis of the liver cannot be expected in so short a time. Nearly two years were required in the previous report.[7] We can therefore only conclude that cirrhosis in the dog cannot be produced in a period of one year by the percutaneous injection of quartz flour into the splenic pulp. It seems probable that this failure is due to the local irritant action of the flour in the splenic pulp preventing an adequate amount of the quartz to reach the liver.

In this connection it might be mentioned that in earlier experiments dicetyl phosphate (.4 gm.) was used as a scarring agent. This proved very toxic when injected into radicals of the portal vein and caused the death of one animal while others went into shock. It may be that a cirrhotic effect might be achieved by using small doses of this drug.

Portal Pressure. An interesting speculation in these experiments has been the possibility of obtaining an accurate estimate of portal pressure directly from the pulp of the transplanted spleen. The desirability of such a procedure is evident. It would allow simple and frequent pressure determination. It would allow these without anesthesia and without operation.

Our experience indicates that there is considerable variation in the portal pressure of normal dogs. This variation is not only present in different dogs but also in the same dogs on different days and under different conditions. We could find no consistent variation resulting from anesthesia. The measured portal pressure

on our normal dogs (omental or mesentaric vein) varied from 150 to 280 mm. H_2O. These were obtained from the animals at the beginning of our experiments. The animals were merely anesthetized to allow laparotomy and pressure determinations. There was no increase in pressure as the experiments progressed. It must be emphasized that any interpretation of portal pressure elevation must be inspected very carefully. Portal pressure is so labile under normal conditions that the introduction of abnormal situations may not cause the elevations attributed to them.

In the splenic pulp pressure determinations of the present study the usual manometer was used. (Fig. 3.) An accurate reading was considered one in which the water steadily fell to what was considered the splenic pulp pressure. At this point there was a fluctuation of a few millimeters with the animals' respirations.

It was thought that the pressures thus obtained were not actually portal pressures. They were splenic pulp pressures which, after all, reflected and were dependent upon portal pressures. An occasional error occurred when the pressure needle impinged against a connective tissue trabeculation or scar within the spleen. When this happened the pressure reached unexplainable heights but was easily remedied by slight manipulations of the needle. Another error occurred several times when the needle point was pushed through the splenic capsule into a subcutaneous position. One animal was lost by intraperitoneal hemorrhage when the needle apparently was run through the hilum of the spleen and injured one of the large vessels.

The results of this phase of the study indicated a fair relationship between pressures taken through the open abdomen and those from the splenic pulp. In general, the splenic pulp pressures were higher (25 to 100 mm.) than those obtained directly from the portal radicles. This may be due in part to the somewhat higher pressure of the closed abdomen. Nevertheless, the pulp pressure followed the same general pressure range of the portal radicles.

It was considered that this means of portal pressure determination was accurate enough for the problem at hand since only broad changes are under consideration. We are certain that for determining slight pressure changes which occur from drugs or transient physiologic effects, portal pressures must still be obtained from the open abdomen adjusted to the portal vein as a base line.

SUMMARY

The spleens of experimental animals were transfixed beneath the skin to facilitate the study of portal pressures and cirrhosis. It was hoped that a fibrosing agent could be introduced indirectly into the portal system by percutaneous injection and that this might result in cirrhosis with portal hypertension. The fibrosing agent used was silicon dioxide. The method failed in part at least, because the quartz caused fibrosis and occasional infection of the subcutaneously placed spleen before enough time elapsed for liver damage to occur. There is reason to believe that the particles of quartz reached the liver by the route outlined and that possibly a cirrhosis could be produced by this method using some other fibrosing agent administered over a longer time.

Attempts were made to determine portal pressure through the splenic pulp of the subcutaneous spleen. It was found that a simple rough determination could be obtained in this manner. Accurate measurements must still depend upon the open abdomen with correction to a common base. It is pointed out that there is much variation in the normal portal pressure of the dog and that changes must be interpreted with care. Elevations formerly reported as portal hypertension are quite within the range we found to be normal and physiologic.

ADDENDUM

Since the completion of our experimental work there has appeared in the English literature a very interesting study in which our same procedure was carried out in humans and with the spleen in its normal position. (Atkinson, M. and Sherloch, S. Intrasplenic pressure as index of portal venous pressure. *Lancet*, 1: 1325, 1954.) Percutaneous splenic pulp pressure readings were taken on twenty-eight patients without mishap. The authors claim a closer relationship between the splenic pulp pressure and portal pressure than we are willing to accept in our own trials. We have had no experience with actual patients and at the present time are hesitant to do so.

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The most common cause of hypotension developing during the course of an operation is reduction in the circulating blood volume due to blood loss or to peripheral vasodilatation. In such instances the administration of intravenous fluids and/or blood transfusions and injection of a vasopressor drug are indicated. However, in a few cases this hypotension is associated with abnormalities of cardiac rate, rhythm and conductivity (caused by auricular flutter or fibrillation, supraventricular or ventricular tachycardia, or atrioventricular block). In these latter instances it might be fatal to overload the venous circulation with excess fluids. (*Richard A. Leonardo, M.D.*)

White Bile in the Common Bile Duct

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ACHOLIA denotes the absence of bile from the intestinal tract. This is usually due to obstruction within the bile ducts. In some cases liver cells fail to produce or to excrete bile. In this report "acholia" is considered as that state wherein (liver) bile does not appear in the bile ducts.

Acholia and the associated liver failure is recognized by the surgeon and the pathologist. It may not be identified as a specific clinical entity by the physician (gastroenterologist). The presence of "white bile" or the absence of bile in the bile ducts is usually of very poor prognosis. Since we have more information today concerning liver function and since no case reports relating to this have appeared in the Index Medicus since 1930, it is thought worth while to review several cases in which "acholia" occurred. These patients are clinically well and fully recovered.

CASE REPORTS

CASE 1. B. S., a sixty-four year old man, was admitted to the hospital on April 20, 1944, because of abdominal pain of six months' duration and jaundice of one month's duration. Diabetes was controlled by diet. The patient had lost 15 pounds during the preceding three years. Ten days before admission the urine was dark-colored, while bowel movements were very light in color.

Physical examination revealed marked jaundice. Liver edge was palpable. A small mass was felt just below the liver edge in the gallbladder area.

Clinical and laboratory examinations included x-rays of the intestinal and biliary tracts. It was concluded that surgical relief of the biliary obstruction was indicated.

Operation was performed on May 1, 1944. There was no evidence of primary or metastatic lymphatic or visceral disease. Positive findings were limited to the pancreatic, biliary and hepatic systems. The pancreas was enlarged, firm and indurated. The gallbladder and common duct were dilated; stones were present in the common duct and gallbladder. The liver was enlarged, greenish and firm.

Clear, slightly pink fluid was in the bile passages. Stones were removed. The common duct and the hepatic duct radicles were irrigated until clear; the papilla of Vater was patulous to injected fluids. Cholecystostomy and choledochostomy were performed. Clear fluid was draining from the T-tube as the operation was completed.

Biopsies of the bile ducts were all benign. The patient's postoperative course was satisfactory except that no bile drained through the T-tube. There was a daily drainage of 30 to 60 cc. of clear colorless fluid for about three weeks. The maximum quantity of cholic acid in this drainage was 50 mg. per 100 cc. Tests for bile pigment, acids, cholesterol and esters were negative. This continued until May 20th. Thereafter, bile color, volume and appearance were normal.

During this three-week period of acholia, which persisted even though the common bile duct was drained, repeated laboratory studies were performed.

Urinalyses revealed that specific gravity ranged from 1.012 to 1.022. A trace of albumin was present occasionally. Bile was present only preoperatively. There was only a trace of urobilinogen present in the undiluted urine, preoperatively. It was not recognized as significant at that time. (Table I.)

Blood counts were normal except that slight leukocytosis occurred during the postoperative acholia. Differential smears were normal. Platelets were normal. (Table II.)

Blood sugar was fairly well stabilized. There was no significant change in the blood urea nitrogen (14 to 21 mg. per cent). Plasma

chlorides and bicarbonate levels were unaltered. (Table III.)

Certain liver function tests were abnormal; the van den Bergh test was elevated preoperatively. After operation it was found that the serum bilirubin level increased during the period when normal bile was absent. Almost within twenty-four hours, after re-establishment of normal color to the bile, the serum bilirubin returned to normal. It was at 0.3 mg. thereafter. (Table IV.)

Prothrombin time was low preoperatively. It remained low until after the restoration of normal bile flow and was normal afterward.

Cholesterol esters were increased from 42 per cent to 64 per cent during the period of acholia.

A significant observation is related to the serum proteins. Total proteins were not altered. However, after an initial drop the globulin partition increased while the albumin fraction decreased. This affected a reversal of albumin to globulin ratio. This appeared after two weeks of acholia. The reversal of ratio continued for seven to ten days after bile flow reappeared. (Table V.)

The alkaline and acid phosphatases showed no significant changes. Abnormalities regressed after normal bile flow was re-established.

Later studies indicated that the bile ducts were not adequate; cholangiogram revealed a long stricture at the distal segment of the common bile duct. Reoperation was performed on June 18, 1944. At this time the common bile duct was anastomosed to the duodenum (end-to-side) in order to maintain free flow of bile into the intestines.

Convalescence was thereafter uneventful. The patient was discharged from the hospital on July 11, 1944.

Follow-up examination in September, 1952, found the patient well and free of any evidence of biliary tract disease.

Case II. Mrs. McS., a thirty-two year old woman, was admitted to the hospital on December 1, 1951, because of severe abdominal pain of one month's duration. Two weeks prior to admission her stools were lighter than normal color.

Cholecystogram had been taken ten days before admission. There was poor function; the gallbladder was dilated and contained many calculi.

She had had a similar attack two years previously accompanied by jaundiced scleras, clay-colored stools and dark-colored urine.

TABLE I
URINALYSES

Urine	4/21	5/4	5/19	5/25
Specific gravity	1.020	1.022	1.012	1.018
Albumin (mg.)	10	75	0	10
Bile	plus 4	negative	.....	.....
Urobilin	trace	.....	.....	.....

TABLE II
BLOOD CELLS

	4/21	5/4	5/19	5/25
Red blood cells	4.91	5.02	5.20	4.47
White blood cells	4.6	6.1	14.4	10.6

TABLE III
BLOOD CHEMISTRY

(mg. vol. %)	4/21	5/4	5/19	5/25
Sugar	115	93	131	139
Urea nitrogen	14	19	11	12
Chloride	620	604	...	560
CO_2	54	50	43	50

TABLE IV
LIVER FUNCTION

Test	4/21	5/4	5/19	5/25
van den Bergh (mg.)	8.0	15.0	20.0	1.0
Prothrombin (Quick)	45%	35%	45%	65%
Cholesterol (mg.)	300	....	278	....
Cholesterol ester	128	....	176	....
Cholesterol ester %	42	....	64	....

TABLE V
SERUM PROTEINS

(Gm. %)	4/21	5/4	5/19	5/25
Total proteins	7.68	6.16	7.04	6.72
Albumin	5.18	4.32	3.42	3.25
Globulin	2.50	1.84	3.62	3.47
A:G ratio	1:2.1	1:2.3	1:0.9	1:0.9

FIG. 1. Case 11. Cholangiogram taken two weeks postoperatively. No calculi. Dye enters duodenum readily. Tiny caliber of common bile duct is visualized as compared to excessive dilatation of common hepatic duct. Intrahepatic ducts normal.

FIG. 2. Case 11. Postoperative cholangiogram. Interductal fistula at papilla of Vater produced at operation by trauma to interductal septum. This permits interductal reflux to occur.

Physical examination revealed slight icterus. A tense tender mass was present in the gallbladder region.

Blood count was normal except for a 12,000 leukocytosis.

Urine concentration was normal. Bile was present. Urobilinogen was found once in dilution of 1:40. Unfortunately, this was not rechecked.

Blood urea nitrogen was 9 mg. per 100 cc. Serum amylase was 49 (Somygyi) units. Icterus index was 40.

Preoperative diagnosis was hydrops of the gallbladder secondary to chronic calculous cholecystitis. It was suspected that choledocholithiasis was present.

The patient was prepared by enteral and parenteral feedings and antibiotics for five days.

Operation was performed on December 6, 1951. A subsiding cholecystitis was present. The inflammation had involved adjacent viscera. Cholelithiasis and choledocholithiasis were present. The gallbladder and common hepatic duct were greatly dilated. The wall of the common bile duct was edematous and thick. The lumen was very narrow being less than 4 to 5 mm. Bile was not in the ducts; only a clear mucoid secretion was found.

A calculus was impacted at the papilla of Vater. It was removed by manipulation and fragmentation using transduodenal and choledochal approaches. This maneuver was known to have injured the septum between the terminations of the common bile and pancreatic ducts.

Cholecystectomy was performed. The common duct was drained by a T-tube. Postoperatively, only a small amount of clear fluid drained through the T-tube. It was noted that the patient was more jaundiced. After seventy-two hours the jaundice began to subside. The bile then returned to normal character. There were no postoperative complications. Cholangiogram was taken on the fourteenth postoperative day. (Fig. 1.) "Dye enters the duodenum without delay. No stones are visualized. The common bile duct is very narrow. The common hepatic duct is dilated. The intrahepatic ducts are normal. A traumatic fistula is present between the terminations of the common bile and pancreatic ducts." (Fig. 2.)

There were no clinical symptoms associated with this interductal reflux. The patient left

the hospital after three weeks. The T-tube was removed after four months.[15] She was readmitted to the hospital on June 18, 1952, because of jaundice. At operation side-to-side anastomosis (hepatic duct-duodenostomy) was performed to permit adequate bile flow. The common bile duct was the site of a lengthy (inflammatory) stricture. No tumor was present.

Convalescence from this operation was uneventful. The tube was permitted to remain as a splint for the anastomosis for six months. At present the patient is well; she has gained over 50 pounds.

COMMENTS

The hepatic failure with acholia reported herein occurred in patients with chronic obstruction of the bile ducts. Common duct stones and infection were present. There was no malignancy.

Obstruction to bile flow produced by a calculus first produces an increase in size and capacity of the duct system.[12] There is, depending upon the degree of obstruction, concurrent increase in intracholedochal pressure. When this exceeds 300 mm. of bile pressure the hepatic cell may not function.[4,6]

Experimental evidence shows that the bile duct epithelium absorbs and excretes, dilutes and depletes the bile which cannot be emptied.[1,11,16] This fluid, except for calcium, becomes nearly isotonic and colorless and contains a good quantity of mucus from intramural duct glands.[2,5] Bile pigment which is present changes from bilirubin through biliverdin to bilicyanin which is colorless.

Acholia most frequently occurs in long-standing cases of obstructive jaundice, occasionally in calculous disease and usually in patients with malignancy.[3,9,10] There is a similar "obstructive phase" in some 20 per cent of patients with hepatitis. Steigmann and Popper[14] studied this problem carefully; they were unable to explain satisfactorily the acholia in such cases. One theory is that exudate may block the periportal region at the junction of bile capillaries and ducts. Another thought has been a rupture of the "canal of Hering"; this simulated the "regurgitation-reflux" mechanism.[7,8,17]

Today the best way to evaluate bile production and flow is in the serial determinations of serum bilirubin; fecal and urinary bile and urobilinogen and through selected liver function tests. The importance of repeated or serial determinations should be stressed.[13]

If the serum bilirubin is found to be stabilized or is decreasing in the presence of obstructive jaundice this should be followed by the disappearance of urine bile, the appearance of bile in the stool, and the presence of fecal and urinary urobilinogen. Should urobilinogen not appear to normal level in the presence of the drop in the icterus index, then it should be suspected that acholia associated with liver failure exists. This indicates that urgent steps should be taken to relieve the obstruction. Such patients should be treated: (1) with antibiotics, (2) by adequate protein and carbohydrate therapy, and (3) by surgical drainage of the bile passages with permanent relief of the obstruction if possible.

Whether or not the bile is permitted to reach the duodenum is not as important as the relief of the obstruction.[14] The liver cell can return to normal function after the causative mechanism originally producing the obstruction is relieved. A secondary operation to reconstruct a proper route for bile flow may be performed at a later elective date.

CONCLUSIONS

1. Acholia has been considered as that state wherein (liver) bile does not appear in the bile ducts.

2. Acholia can occur in the presence of obstructive jaundice whether it be inflammatory (cholangitis or hepatitis), calculous or neoplastic.

3. Presence of white bile in the common duct (or the absence of bile therein) indicates hepatic insufficiency. This usually prophesies a subsequent fatality.

4. Acholia does not necessarily carry a poor prognosis in patients who have benign obstruction to the ducts or in those who have hepatitis.

5. It is important to correlate fecal and urine urobilinogen with serum bilirubin levels over consecutive days in order to determine, for example, that a lowered serum bilirubin level actually is associated with the appearance of bile in the intestinal tract.

6. An indication for early surgical intervention in a patient with obstructive jaundice exists when the serum bilirubin is stabilized or decreasing in the absence of positive proof that bile is reaching the intestinal tract.

7. Drainage of the bile passages under such

conditions is essential in order to permit an opportunity for the hepatic cel function to be restored.

SUMMARY

Two patients are reported who had chronic calculous obstruction to the common bile duct. They recovered from a severe form of hepatic insufficiency manifest by "acholia." The necessity for suspecting the presence of this type of liver failure is emphasized.

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Treatment of Viral Hepatitis with the Knott Technic of Blood Irradiation

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The results of treatment of forty-three patients with viral hepatitis by the Knott technic of blood irradiation therapy are presented. Thirty-one cases of acute infectious hepatitis are included in this series; three cases were chronic and of long standing, and twelve were cases of acute serum hepatitis. These patients received no therapy other than the Knott technic of blood irradiation. The average follow-up period was 3.56 years. Twenty-two patients were followed up for four to six years, seventeen for one to three years and four for two to six months.

The following criteria, considered by Stauffer[1] as essential for positive diagnosis of viral hepatitis, were employed: Thymol turbidity (within two weeks of onset of jaundice), above 10 units; cephalin flocculation (within two weeks of onset of jaundice), 4 plus; urinary urobilinogen, over 1:40 dilution; serum bilirubin, above 1.2 mg. per cent.

RATIONALE AND METHOD OF TREATMENT

The Knott technic of blood irradiation (approved by the American Blood Irradiation Society) achieves the following physiologic objectives: (1) increases the blood oxygen level;[2,3] (2) increases phagocytosis,[4–6] (3) relieves toxemia;[7,8] (4) decreases edema;[7,9] and (5) controls nausea and vomiting.[9]

Treatment consists of withdrawing 1.5 cc. of blood per pound of body weight from the patient, citrating it and, by use of the Knott hemo-irradiator, exposing it to radiant energy between the wave lengths of 2,399 and 3,900 angstrom units as it passes through the irradiation unit at a predetermined rate. The blood is returned to the patient through the needle used for the initial venipuncture. Treatment requires from fifteen to twenty minutes. Outpatients rest fifteen minutes, after which time they may resume whatever activity is permitted.

RESULTS OF TREATMENT

Tables I and II list the forty-three cases covered by this report, indicating age, race and sex of patient, diagnosis, number of blood irradiation treatments administered, number of days of illness after irradiation, recurrences and follow-up period.

An average of 3.28 treatments per patient were administered in this series. Laboratory studies were employed to confirm clinical improvement, which occurred on an average of 19.2 days after institution of blood irradiation therapy. Sixty per cent of the patients were considered clinically recovered and able to return to their occupations in two weeks or less.

CASE REPORTS

The following cases have been chosen as typical of those presented in this series:

Case 15. F. M., a twenty-eight-year old white man had complained of anorexia, nausea and pain in the right upper quadrant for three weeks. When first seen he was deeply jaundiced. Laboratory findings were as follows: Icteric index, 54 units; thymol turbidity, 15 units; cephalin flocculation, 4 plus; urinary urobilinogen in excess of 1:40 dilution and serum bilirubin in excess of high normal.

The patient was given one blood irradiation and discharged from the hospital ten days later, clinically free of symptoms. All laboratory findings returned to normal at the end of two weeks. Patient has had no recurrences during the five-year follow-up period.

Case 16. M. O., a thirty-eight year old white woman, gave a history of having clay-colored stools, very dark urine and yellow

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TABLE 1

SERUM HEPATITIS*

Case No.	Age, Race and Sex	Diagnosis	No. of Blood Irradiations Administered	Clinical Impressions and Remarks	No. of Days to Complete Recovery after Blood Irradiation	Recurrences	Follow-up Period
1.	29, W, M	Acute, severe	6	Improvement noted after first irradiation; patient discharged from hospital in 7 days much improved	13	None	3 yr.
2.	19, W, M	Acute, mild	1	Improvement in 2 days	7	None	6 yr.
3.	44, W, M	Acute, moderately severe	1	Improvement noted 3 days after blood irradiation	18	None	2½ yr.
4.	35, W, F	Acute, severe	2	Patient ill 10 days before treatment; improvement noted in 24 hr.; marked improvement in 4 days	14	None	1 yr.
5.	24, W, F	Acute, very severe	3	Patient ill 6 wk. prior to treatment; marked improvement in 3 days after first blood irradiation; discharged from hospital in 13 days	14	None	4 yr.
6.	40, W, M	Acute, moderately severe	2	Marked improvement in 2 days	7	None	3½ yr.
7.	52, W, M	Acute, very severe	2	Marked improvement 7 days; discharged from hospital completely recovered in 14 days; patient subsequently had surgery and 52 transfusions over 6 mo. period with no recurrence of serum hepatitis	14	None	5 yr.
8.	40, W, F	Acute, moderately severe	2	Patient presented complication of renal insufficiency; marked improvement in 48 hr.	18	None	4 yr.
9.	45, W, M	Acute, severe	5	Nausea disappeared after first blood irradiation; appetite improved, jaundice disappeared after second	14	None	2 mo.
10.	19, W, M	Acute, moderately severe	3	Improvement noted after first blood irradiation; patient was heroin addict and reinfection rather than recurrence was suspected	14	One, possible reinfection	1 yr.
11.	56, W, M	Acute, severe	2	Marked improvement noted in 2 wk.	30	None	3½ yr.
12.	48, W, M	Acute, severe	3	Patient ill 6 wk. prior to blood irradiation without improvement; improvement noted 1 wk. from first irradiation	42	None	5 yr.

* Serum hepatitis occurs following some injections, such as blood plasma, etc.

TABLE II

INFECTIOUS HEPATITIS*

Case No.	Age, Race and Sex	Diagnosis	No. of Blood Irradiations Administered	Clinical Impressions and Remarks	No. of Days to Complete Recovery after Blood Irradiation	Recurrences	Follow-up Period
13.	40, C, M	Acute, moderately severe	1	Serum bilirubin dropped from 11 to 2 in 7 days; patient returned to work as laborer in 14 days	14	None	4 yr.
14.	32, W, F	Acute, moderately severe	1	Improvement noted in 3 days	9	None	6 yr.
15.	28, W, M	Acute, moderately severe	1	Improvement noted in 3 days	10	None	5 yr.
16.	38, W, F	Acute, postinfluenzal, very severe	4	Improvement noted after first irradiation	7	None	3½ yr.
17.	14, W, M	Acute, moderately severe	3	Discharged from hospital much improved in 4 days	10	None	3 yr.
18.	46, W, M	Acute, very severe	6	Steady improvement after first irradiation	35	None	5 yr.
19.	18, W, F	Acute, mild	1	Improvement in 2 days	7	None	6 yr.
20.	50, W, M	Acute, mild	1	Marked improvement in 2 days after blood irradiation	7	None	4½ yr.
21.	35, W, F	Acute, moderately severe	2	Marked improvement in 2 days	8	None	5 yr.
22.	42, W, M	Acute, mild	1	Marked improvement day after blood irradiation	7	None	5 yr.
23.	40, W, F	Acute, severe	3	Improvement in 7 days; recovery slow but uneventful	49	None	5½ yr.
24.	8, W, F	Acute, mild	1	Improvement in 2 days	7	None	5 yr.
25.	27, W, M	Acute, very severe	5	Marked improvement in 6 days	28	None	3½ yr.
26.	24, W, F	Acute, moderately severe	3	Marked improvement in 2 days	7	None	3½ yr.
27.	8, W, F	Acute, mild	1	Marked improvement in 2 days	7	None	5 yr.
28.	19, W, M	Acute, severe	3	Some improvement 3 days; marked improvement 7 days	14	None	4 yr.
29.	10, W, F	Acute, mild	1	Marked improvement 24 hr.	7	None	2 yr.

* Infectious hepatitis occurs following ingestion of contaminated water or food and is encountered most frequently in Army posts, schools, camps, etc.

TABLE II (*Continued*)

Case No.	Age, Race and Sex	Diagnosis	No. of Blood Irradiations Administered	Clinical Impressions and Remarks	No. of Days to Complete Recovery after Blood Irradiation	Recurrences	Follow-up Period
30.	20, W, F	Acute, moderately severe	2	Marked improvement 11 days	21	None	5 yr.
31.	65, W, M	Acute, severe	2	Some improvement 3 days; marked improvement 6 days	14	None	2 yr.
32.	31, W, F	Acute, very severe	4	Patient ill 3 wk. before treatment; marked improvement 2 days; discharged from hospital ambulatory; temperature normal in 4 days	16	None	4½ yr.
33.	14, W, M	Acute, very severe	4	Patient ill 9 days before treatment; marked improvement in 5 days; discharged from hospital in 10 days	42	None	3 mo.
34.	43, W, F	Acute, very severe	6	Patient ill 4 mo.; did not respond to conventional management; marked improvement 3 wk. after first irradiation	90	None	2 yr.
35.	41, W, M	Acute, very severe	4	Patient ill 2 wk. before treatment; improvement noted 3 days after first treatment; discharged from hospital in 19 days	21	None	6 mo.
36.	35, W, F	Acute, severe	5	Marked improvement 1 wk.	14	None	2 yr.
37.	51, W, F	Acute, severe	6	Progressive improvement to complete recovery after first treatment	14	None	2 yr.
38.	27, W, M	Acute, very severe	6	Patient ill 4½ mo. prior to treatment; marked improvement 8 days after first blood irradiation	17	None	2 yr.
39.	29, W, M	Acute, moderately severe	2	Patient gave history of initial attack in 1943, second in 1944, third in 1945; first blood irradiation administration in 1947; patient recovered in 7 days; recurrence in 1948, blood irradiation again administered, followed by recovery in 7 days; no further recurrences	7	One after first irradiation, none after second	6 yr.

TABLE II (*Continued*)

Case No.	Age, Race and Sex	Diagnosis	No. of Blood Irradiations Administered	Clinical Impressions and Remarks	No. of Days to Complete Recovery after Blood Irradiation	Recurrences	Follow-up Period
40.	26, W, M	Acute, very severe	6	Patient gave history of initial attack in 1946 with three recurrences by 1950, at which time he received blood irradiation; improvement noted 5 days after first treatment	22	None	4½ yr.
41.	49, W, F	Acute, very severe	5	Improvement after second blood irradiation; jaundice disappeared after third	28	None	4 mo.
42.	35, W, M	Acute, very severe	11	Improvement noted after first blood irradiation	38	None	2 yr.
43.	26, W, M	Acute, very severe	8	Patient gave history of chronic viral hepatitis of 4 yr. duration during which he experienced three attacks; patient had been ill for 1 yr. before blood irradiation; some improvement after first irradiation, much improved after third	45	None	6 yr.

scleras of one week's duration, with anorexia and nausea of two weeks' duration and weight loss of 5 pounds in the thirty-day period preceding examination. There had been no abdominal pain either before or after the onset of symptoms.

Examination revealed the following: icteric tinge to scleras in eyes, tongue shiny and coated, gingiva somewhat icteric, and skin pale lemon yellow tinge. The liver, kidney and spleen were not palpable; no tenderness was elicited to superficial or deep pressure. Moderate tympanitis was present. There was no distention and bowel sounds were normal.

Laboratory findings gave the following data: Icteric index, 36; van den Bergh test: direct, positive; indirect, 3.8; total proteins 7.0; serum albumin, 3.9; serum globulin, 3.81; the A.G. ratio was, therefore, 1:1; Kahn test, negative; cephalin flocculation, 4 plus positive.

Blood irradiation by the Knott technic was administered. Icteric index dropped to 16 at the end of twenty-four hours. Three more blood irradiations were administered at two-day intervals, at the end of which time the icteric index had dropped to 6.

The patient was asymptomatic eight days following the first blood irradiation, at which time laboratory studies confirmed clinical recovery. During the subsequent four-year follow-up period, the patient has had no recurrences.

Chronic cases of viral hepatitis, while responding more slowly, appear to benefit definitely from blood irradiation therapy. The following is a report of a chronic case of four years' duration, the diagnosis of which was later confirmed when surgery was performed, revealing "scars" on the liver:

CASE 43. H. A. Z., a twenty-six year old white man, gave a history of first appearance

symptoms in April, 1944, while on duty with the Army, at which time abdominal cramps, vomiting and diarrhea developed. This episode lasted five or six days, with fifteen to eighteen bowel movements daily; there was no blood in the stools. The patient was given belladonna.

Symptoms of vomiting, diarrhea and severe abdominal pains reappeared in July, 1945, accompanied by jaundice. Diagnosis was infectious jaundice and the patient was hospitalized for twenty-one days. Treatment consisted of a low fat, high protein and carbohydrate diet and bedrest.

The patient was discharged from the Army in January, 1946, at which time he weighed 150 pounds. He began to notice weakness with occasional vomiting in December, 1946. One month later he began having severe vomiting episodes and severe cramping abdominal pains in the right upper quadrant; no blood was vomited, nor was any blood present in stools. He was hospitalized at a veterans hospital three times the following year with varying diagnoses of duodenal ulcer and cirrhosis of the liver. However, at no time were these diagnoses confirmed.

Symptoms continued throughout 1947 and to May, 1948, at which time his weight had dropped from 150 to 117 pounds. Morphine was used parenterally to relieve severe burning pains in right upper quadrant, associated with severe episodes of vomiting.

The first blood irradiation was administered by the Knott technic on May 1, 1948, after laboratory studies had confirmed the clinical diagnosis of infectious hepatitis. A second blood irradiation was given one week later, at which time the patient reported having slept well since the previous irradiation, with no attacks of pain or vomiting (which he had been experiencing almost daily prior to the first blood irradiation). There was an 8 pound weight gain. A third blood irradiation was administered on May 15th, at which time the patient showed no evidence of relapse and had a weight gain to 130½ pounds. A fourth blood irradiation was given on June 15th, at which time the patient had gained a total of 18½ pounds and laboratory studies had returned to normal.

Follow-up examination on October 19th revealed the patient's weight to be 144 pounds, a total gain of 27 pounds. Re-examination four months later revealed no further attacks; maintenance of the weight gained and laboratory studies confirmed clinical recovery.

On April 14, 1949, the patient showed indications of relapse, at which time blood irradiation therapy was again administered, although laboratory studies failed to confirm the existence of an acute attack of infectious hepatitis; cephalin flocculation was 2 plus, bromsulphalein in five minutes, 52 per cent, thirty minutes 13 per cent. Patient was again given irradiation on May 9, 1949, and was hospitalized on May 24th because of recurrence of severe pain in right upper quadrant. X-ray studies revealed spasm of the duodenal sphincter and surgery was instituted because of the possibility of organic disease beyond the existence of chronic, active viral hepatitis. Operation consisted of: (1) adhesolysis, (2) Ramstedt pyloroplasty and (3) appendectomy.

Findings at operation revealed the following: Liver was not enlarged, margins were smooth, and numerous linear surface scars measuring 1 to 1½ cm. covered its anterior surface, associated with many small, yellowish placques. The gallbladder was dilated and surrounded with numerous adhesions in the region of Hartmann's pouch and the cystic duct. The pylorus appeared somewhat thickened but there was no evidence of any recent or pre-existent ulcer. No biopsy of the liver was made because inflammatory changes appeared to be nonspecific and quiescent.

The patient recovered uneventfully and has had no further recurrences of viral hepatitis and no disability of any type.

PREVENTION OF RECURRENCES

Statistics with regard to the frequency of relapse in viral hepatitis vary from 0.6 to 18 per cent,[10] although literature in the last two years places the incidence of recurrence somewhat higher than in previous years and at an average of approximately 10 per cent. The prevention of even a small percentage of recurrences, however, is a vital consideration in the treatment of this disease since permanent liver damage is frequently the result of recurrences.

In this series there were two recurrences, or an average of 4.65 per cent. One recurrence (Case 39) occurred in a patient with a history of chronic infectious hepatitis of four year's duration, during which time he experienced four attacks. Blood irradiation therapy was

instituted at the time of the fourth attack. The patient recovered in seven days but experienced one relapse subsequently. He was again irradiated and no further attacks occurred during the ensuing six-year follow-up period. The patient in Case 10 was a heroin addict and reinfection, rather than an actual recurrence, was suspected.

COMPARISON WITH OTHER THERAPY

Conventional treatment currently employed in cases of viral hepatitis consists of bedrest and a well balanced, nutritious diet, under which management the average period of illness is from six to eight weeks.[10]

Although aureomycin has been reported to be of some value in patients who are seriously ill or in hepatic coma,[11] none of the chemotherapeutic or antibiotic agents now known appear to exert any beneficial effect on the average case of viral hepatitis.[12]

Reports covering the administration of ACTH to patients in the acute phase of viral hepatitis indicate that while there was a diminution in the amount of serum bilirubin and a slight shortening of the duration of the disease, certain untoward effects were noted, such as "mooning" of the face, ascites, edema and hypertension;[13,14] relapse following too short a course of ACTH therapy was common.[10]

In this series no patient treated by the Knott technic of blood irradiation was found unable to tolerate the therapy, nor were any unfavorable reactions or untoward effects noted. The rapid subsidence of nausea, vomiting, anorexia, pain and jaundice has been a major factor in our observations of results achieved by use of this technic in these cases.

Table III represents a comparison of results obtained by use of the Knott technic of blood irradiation in forty-three cases of viral hepatitis, as contrasted with other therapeutic measures currently employed.

SUMMARY

1. Forty-three patients with acute viral hepatitis were treated with the Knott technic of blood irradiation therapy. Thirty-one of these patients had acute infectious hepatitis, three were chronic and of long standing, and twelve had acute serum hepatitis.

2. An average of 3.28 treatments per patient were administered.

3. The average period of illness after the Knott technic of blood irradiation therapy was instituted was 19.2 days. Clinical recovery was confirmed by laboratory studies.

4. Two recurrences were observed among the forty-three patients treated, or an average of 4.65 per cent during a follow-up period averaging 3.56 years.

5. No deaths occurred among these forty-three patients.

6. A rapid subsidence of symptoms of nausea, vomiting, anorexia, pain and jaundice was noted in all patients treated, as well as a coincident trend back to normal of laboratory studies. Marked improvement was noted in twenty-seven patients within three days or less after institution of blood irradiation therapy. Eleven patients showed marked improvement in four to seven days and five patients were markedly improved in eight to fourteen days.

7. No untoward effects or unfavorable reactions were observed as the result of blood irradiation therapy in these patients and no patient was found unable to tolerate the therapy or to develop a resistance to it.

TABLE III*

	No. of Treatments	Duration of Illness (days)	Recurrences (%)	Deaths (%)
Irradiated patients.....	3.28	19.2	4.65	None
Non-irradiated patients (statistical average[10])	....	42 to 56	0.6 to 18	0.2–19

* Results listed are average.

CONCLUSIONS

It is believed that the Knott technic of blood irradiation therapy can be relied upon to terminate promptly an acute attack of viral hepatitis, to prevent recurrences and to arrest liver damage as a result thereof.

An increase of over 300 per cent in the incidence of viral hepatitis during the past three years is indicated by the following figures furnished by the Department of Health, Education and Welfare, Public Health Service, National Office of Vital Statistics:

	No. of Cases Reported
1951	7,349
1952	17,428
1953	33,382

In the face of the growing problem presented by this disease and the absence of any effective treatment for it, the results presented herein are considered significant and noteworthy.

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Osteosynthesis and the Lane Technic

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OSTEOSYNTHESIS has been of varying but on the whole increasing importance in the treatment of fractures since the time of Lambotte, Lane, König and Murray. In the evaluation of this method of treatment the same mistake is made time and again: good results are expected from bad technic. There is no method of treatment, either conservative or operative, which yields satisfactory results if its application is not correct, unless nature has mercy on the patient and he recovers in spite of the treatment. This, however, can never be the method of choice of the attending physician. It is for this reason that the Lane method of osteosynthesis is repeatedly regarded adversely. The same view was held of conservative treatment using plaster of paris, until the value of its correct application was proved by Böhler.

When a general comparison is made between conservative and operative treatment (osteosynthesis), we must bear in mind that repeated manipulations under heavy traction may cause more severe trauma than a careful, technically justified osteosynthesis.

The indications for osteosynthesis are of great importance. If satisfactory healing of a fracture can be effected by conservative treatment, with good position of the axis, good function of the extremity and normal duration of the healing period, and the general condition permits it, osteosynthesis is not indicated. Osteosynthesis is called for, on the other hand, if reduction or retention in position of the fracture, or both, lead to difficulties. The question still to be answered is whether in a given case osteosynthesis is possible, and what method is the most suitable. This decision should be made immediately after the trauma, or in any case within the first two weeks, so that osteosynthesis can be performed before various more or less radical conservative manipulations have been carried out, and before reconstruction is in full progress.

The following factors have to be taken into account when the indications for osteosynthesis are being considered: (1) The patient should be in good general condition; he must have recovered from shock, if any, and general anesthesia should be possible. (2) The condition of the soft tissues should be healthy; they must show good circulation, innervation and function. The skin should be intact. Great reserve is called for in performing osteosynthesis in the case of an open wound. If there is any doubt regarding healing by first intention, we have to wait for the healing of the wound, usually under a traction dressing. Direct osteosynthesis should never be performed in war casualties. (3) The type of fracture should be such that osteosynthesis is technically feasible. The first demand made on osteosynthesis is anatomic reduction with adequate fixation. This fixation should not be expected to stand weight bearing immediately following the operation. Osteosynthesis serves only to enable the extremity to move freely without a load, if necessary using a traction bandage. If we want to apply a load, extra support is required, such as a plaster of paris dressing or a Thomas splint, this view being contrary to that of Murray who is opposed to any bandaging in the after-treatment.

The method of osteosynthesis should be chosen for each individual case. It is of importance that as little callus-forming tissue as possible be damaged during the operation. The periosteum should therefore be handled very carefully, i.e., the "entity" skin-fascia-muscle-periosteum should be left intact and, when possible, the fracture fragments not skeletized circularly. Adequate measures should be taken against infection. We must therefore aim at an aseptic technic, controlled hematoma and the use of inert fixation material. Ligations are to be avoided and sutures under tension are not allowed. Anatomic reduction is an absolute

demand; thus the soft parts also develop anatomic relationships with good circulation and good function.

Wiggins et al. of the U.S. Naval Hospital in Philadelphia, give in their article, "Complications following open reduction and plating of fractures of the tibia," (*Am. J. Surg.*, 86: 273, 1953) a biased consideration of osteosynthesis according to the Lane method; their judgment is anything but favorable.

The concise historical survey and the indications given by Lane, Murray, McLaughlin, Clarke, Conwell and Harris give a general view of the indications for osteosynthesis and the requirements regarding its performance and technic. On the strength of statistics from the Naval Hospital on 139 fractures of the shaft of the tibia (of which there were fifty-three cases with complications, including eleven after treatment by the Lane osteosynthesis method), the authors believe they are justified in condemning it. They advocate their own ideas, namely, manipulative reductions and plaster of paris in transverse fractures, open reduction and osteosynthesis with screws in oblique fractures, traction and transfixation in closed comminuted fractures and all open fractures.

This, however, is not the proper way of dealing with the problem. Wiggins's Table 1 and his x-ray pictures suggest that it is not the osteosynthesis which is to blame for the bad results, but rather wrong indications, inadequate osteosynthesis and failure to observe asepsis. Under war conditions in advanced areas every form of osteosynthesis should be forbidden. If the indications and technic are correct, cortical fixation with the Lane plate remains an acceptable form of therapy. Another important factor in osteosynthesis is the after treatment. In no case should osteosynthesis be directly exposed to weight bearing. "Unloaded" mobilization favors callus formation. The weight bearing may go so far that there is still no mobilization in the fracture; it may increase as the callus develops. The intelligent patient feels this himself and regulates it spontaneously. Others will need a plaster cast or some sort of apparatus in addition to osteosynthesis during the callus formation.

When viewed from this angle the Lane method will retain its place. It is not fair to expect that a method of treatment will yield good results if its application is incorrect. Even though Wiggins et al. obtained good results by working along their lines, we still cannot accept their method as that of choice.

Each fracture must be dealt with individually, according to the general ideas and technical possibilities. The Lane plate will most certainly have to remain part of our technical armamentarium, and therefore I should like to modify the conclusion of these authors as follows:

"A great need exists for re-evaluation of the indications and technic for osteosynthesis. This is not the innocuous procedure that many surgeons have been led to believe. Osteosynthesis should be reserved for those individuals who have been thoroughly indoctrinated in the basic principles and technic as expounded by Lane and Murray."

Up to 1952 my statistics of osteosynthesis by the Lane method cover 128 cases of fractured tibia, forming 20 per cent of all cases of fracture of the tibial shaft. (Table 1.) The first column mentions the various factors that will be discussed shortly. Columns II to V show the figures referring to the various types of fracture; column VI gives the general number without any distinction regarding the type of fracture. The figures on the left side of the columns refer to the patient. Figures 1 to 9 show the different types of fracture, the osteosynthesis and the consolidation.

The following is a brief discussion of the factors from column 1.

Age. In general, age has no important influence on the results of these statistics. Only if the series under treatment consisted of very youthful cases would the figures be influenced favorably.

Complicated Fractures. There are twenty-five complicated fractures, mostly atypical and transverse.

Day of Osteosynthesis. In 118 cases the indication for osteosynthesis was accepted between the first day and three weeks, and in ten cases between three weeks and six months following the trauma. In the latter case we speak of "late osteosynthesis."

COMPLICATIONS AFTER OSTEOSYNTHESIS

Removal of Plate and Screws

In twenty-eight cases symptoms of irritation necessitated removal of the plate one to twelve years after osteosynthesis. These symptom complexes are: (1) corrosion-metallosis, excess

TABLE I

I	II	III	IV	V	VI
Osteosynthesis in Fractures of Tibia	Atypical (Compound) Fractures (24) (Figs. 1 to 3)	Transverse Fractures (34) (Fig. 4)	Spiral Fractures (54) (Fig. 5)	Oblique Fractures (16) (Fig. 6)	Total (128)
Age:					
15 to 25 yr.	7	11	24	10	52
25 to 50 yr.	14	18	27	5	64
50 yr.	3	5	13	1	22
Open fractures*	9*	12*	2*	2*	25*
Day of osteosynthesis:					
1 to 3 wk.	24	28	50	16	118
3 wk. to 6 mo.	..	6	4	..	10
Anatomic position	20	30	45	15	110
Good	4	4	9	1	18
Complications:					
Removal of plate	5	10	11	2	Bad Alloy (25), Vitallium (3) 28
Fat embolism	No. 44 1	..	..	..	1
Osteitis	..	No. 47* and 36* 2	..	..	2
Slow callus formation	No. 37 Major trauma, No. 45 Major trauma, No. 82 Diastasis 3	No. 24* Diastasis, No. 30 Diastasis, No. 36* Osteitis 3	No. 41 Atrophy, refracture 1	No. 63 Diastasis, refracture 1	Diastasis 4, Major trauma 2, Osteitis 1, Atrophy 1 8
Pseudarthrosis*	No. 26* Double fracture, No. 79* Compound fracture from major trauma 2	No. 47* Osteitis, No. 43b* Late osteosynthesis, Beck's drilling, No. 34* Diastasis, Beck's drilling 3	.....	No. 83 Late osteosynthesis, wrong treatment, Kirschner operation 1	Compound fracture 2, Diastasis 1, Osteitis 1, Late osteosynthesis 2 6
Disability (with slow callus formation and pseudarthrosis)	No. 37 Treatment 6 mo. No. 45 Treatment 8 mo. No. 82 Treatment 6 mo. No. 26* Treatment 13 mo. No. 79* Treatment 12 mo.	No. 24* Treatment 7 mo. No. 30 Treatment 5 mo. No. 36* Treatment 8½ mo. No. 34* Treatment 9 mo. No. 43b* Treatment 16 mo. No. 47* Treatment 4 yr.	No. 41* Treatment 7 mo.	No. 83* Treatment 18 mo.	No lasting disability
Structural callus formation	13 wk.: 19	15 wk.: 28	11 wk.: 53	12 wk.: 14	3 to 4 mo.: 114

* = complicated fracture.

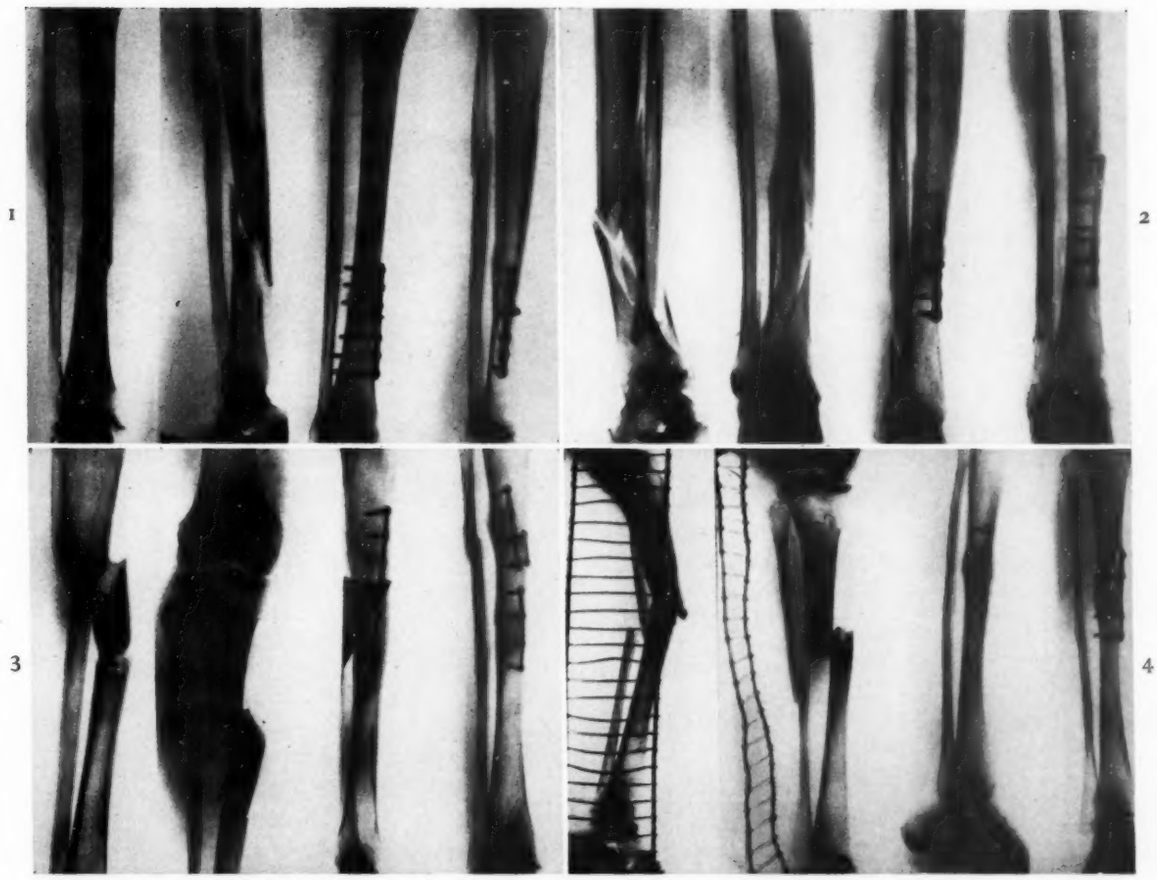


FIG. 1. Atypical (compound) fracture in forty-nine year old man. Lane osteosynthesis performed April 24, 1951. Consolidation occurred June 24, 1951.

FIG. 2. Atypical (compound) fracture. Lane osteosynthesis performed.

FIG. 3. Atypical (double) fracture in thirty-five year old woman. Lane osteosynthesis performed May 11, 1949. Consolidation occurred September 20, 1949.

FIG. 4. Transverse open fracture in a twenty-four year old woman. Lane osteosynthesis performed July 31, 1951. Fat embolism. Normal consolidation occurred November 20, 1951.

callus formation, edema and pain; (2) bad fixation and fracture of plate or screw (corrosion-metallosis, slow callus formation up to pseudarthrosis); and (3) infection and osteitis (corrosion-metallosis, pseudarthrosis).

There is a great difference between vitallium® and the other alloys. The alloys sold as rustless steel, e.g., in the sense of V_2A, are certainly not reliable. Metallurgic examination proves that in a great number of cases the composition is not in agreement with the alloy for which it is sold. Screws and plates may be of different composition. A check on the hardness and toughness of the material also gives disappointing results. Corrosion and metallosis were not observed with the use of vitallium. Up to the time of writing we have had to remove a plate in only three cases, the causes being overloading, bad fixation or osteitis. In these cases we do not blame the method for the bad results, but the technic and the faulty indications.

The symptom complex of corrosion-metallosis, excess callus formation, edema and pain may occur from one to ten years or more after osteosynthesis. It is important that an inert fixation material be used, for which purpose vitallium is excellent. Removal of the Lane plate does not give rise to any difficulty. In the case of the previously mentioned symptom complex, the patient can go to work again after three to four weeks.

Fat Embolism

Fat embolism was observed once in a complicated fracture after direct osteosynthesis. (Fig. 4, Patient No. 44.) This patient was

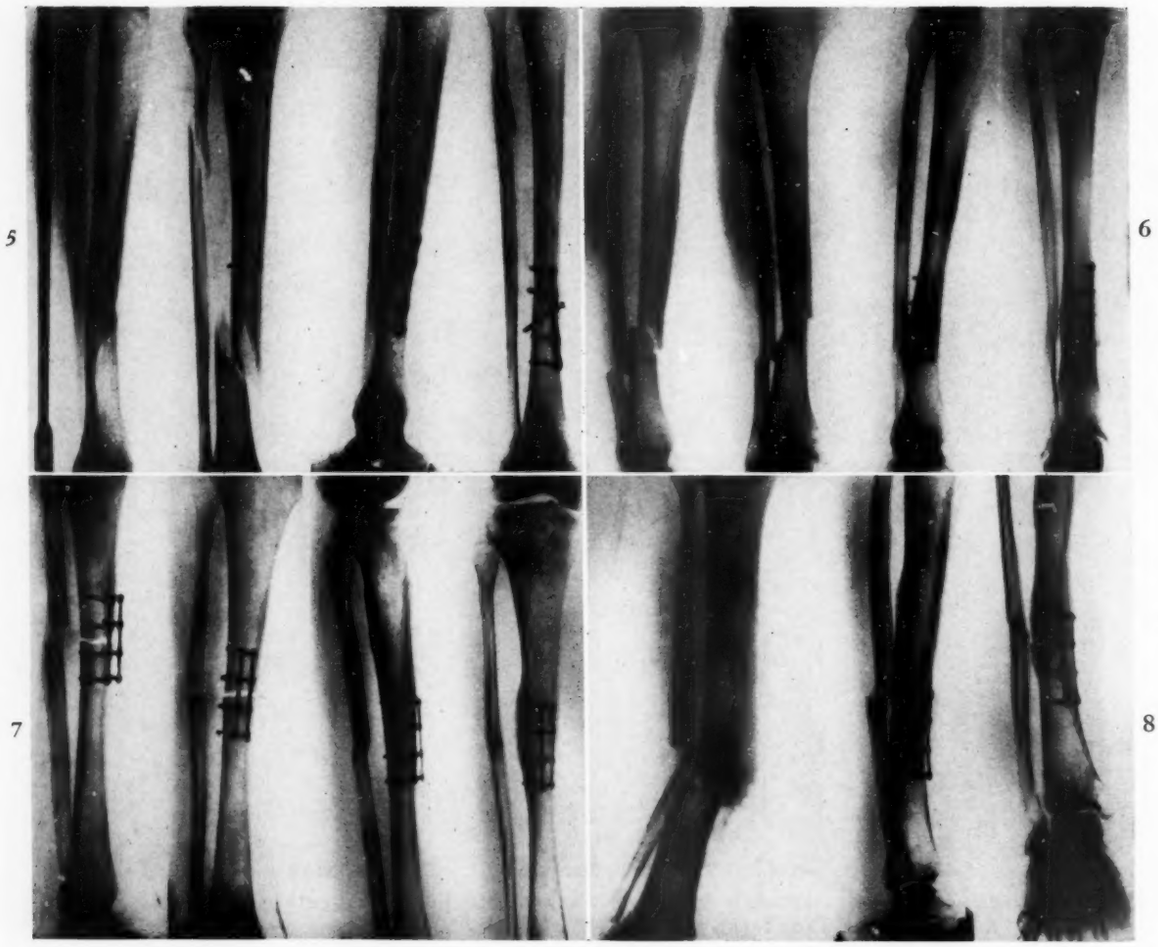


Fig. 5. Spiral fracture in seventy-three year old man. Lane osteosynthesis performed August 29, 1951. Consolidation occurred October 20, 1951.

Fig. 6. Oblique fracture in a twenty-one year old man. Lane osteosynthesis performed October 15, 1948. Consolidation occurred December 21, 1948.

Fig. 7. Open fracture in fifty year old man. Diastasis. Lane osteosynthesis performed September 11, 1948. Consolidation occurred March 8, 1949.

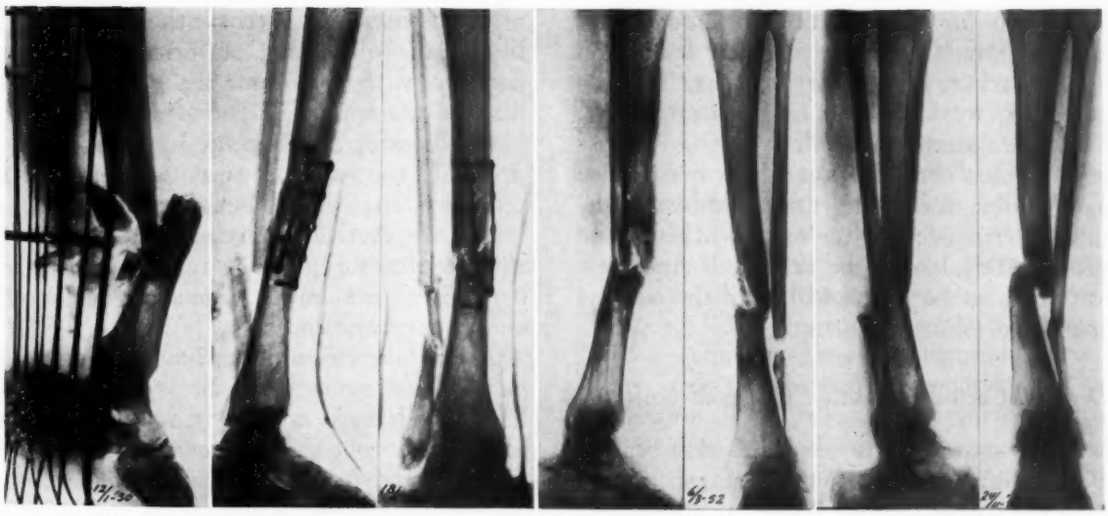
Fig. 8. Open fracture in forty year old man. Proximal fragment protrudes 2 inches from wound, which measured 7 inches. Laceration of the muscles. Lane osteosynthesis performed August 11, 1948. Consolidation occurred January 11, 1949. No disablement on April 25, 1950.

admitted to the hospital in bad general condition (shock) with a compound fracture and a large wound. There was an undisturbed recovery, both of the fracture and the serious clinical picture of fat embolism. We do not know whether the osteosynthesis *per se* had a favorable influence in this serious condition.

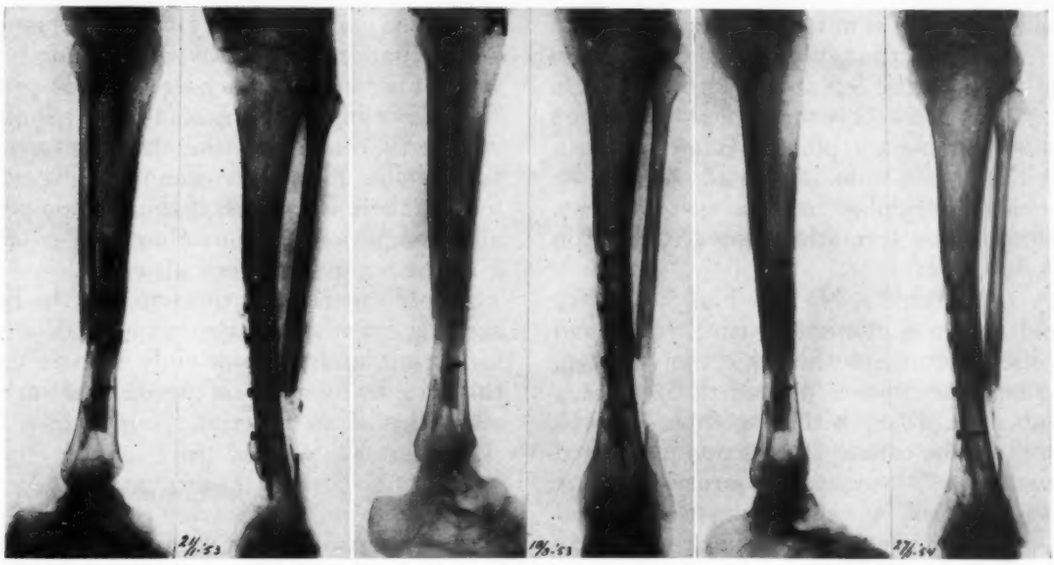
Osteitis

Case 1. (Table 1, No. 47, Fig. 9.) This forty-one year old man was admitted with a serious transverse compound fracture. The tibia protruded from the soft tissues for a distance of 20 cm., with complete detachment of the periosteum. The wound was lacerated and full of coal dust. Amputation was contemplated but not performed.

On January 12, 1950, the day of the accident, a Lane osteosynthesis was performed within six hours, after débridement of the wound. To close this relief incisions were necessary on the medial and lateral sides which were repaired by means of pinch grafts one week later. Wound healing was uneventful. On May 23, 1950, the patient was put in a walking plaster. On September 5, 1950, symptoms of osteitis arose



9A 9B 9C 9D



9E 9F 9G

FIGS. 9A to D. A, open fracture. B, osteosynthesis. C, osteitis; removal of plate and screws on September 5, 1950. D, pseudarthrosis with constructive bone formation.

FIGS. 9E to G. E, grafting. F, check-up. G, consolidation; patient resumed work.

with fistula formation in extensive scar tissue. The vitallium Lane plate and screws were removed.

The first attempt at skin plasty was made on March 6, 1951. It was not until January 15, 1953, that all signs and symptoms of osteitis had disappeared and the condition of skin and soft tissues permitted a pseudarthrosis operation. During the operation the sclerotic tissue was removed, the medullary space widely opened, the bone fragments repositioned anatomically and fixed with a bone graft from the other tibia by means of vitallium screws in a graft bed made beforehand.

On January 14, 1954, there was constructive consolidation in an anatomic position, with normal function of the extremity.

CASE 11. (Table 1, No. 36.) A thirty-two year old man was admitted with a compound transverse fracture of the tibia and fibula and multiple abrasions of the skin.

On March 29, 1951, the day of the accident, a Lane osteosynthesis with a vitallium plate and screws was performed within six hours. A mild infection arose and the Lane plate and screws were removed on July 2, 1951. There was neither metallosis nor corrosion. Treatment was continued with a circular plaster cast

extending as far as a handbreadth below the inguinal region. Constructive callus formation was observed on November 29, 1951. The patient went to work again on December 14, 1951.

These two cases show that osteitis constitutes a serious complication which may cause delayed callus formation and pseudarthrosis. Should osteitis occur after osteosynthesis, the fixation material must be removed. Any new operation must be postponed until the osteitis has subsided (Watson Jones).

Delayed Callus Formation (Delayed Union)

Table 1 shows eight cases of slow callus formation, four of them due to diastasis during the osteosynthesis.

CASE I. (Table 1, No. 82.) A twenty-three year old man was admitted with a compound fracture with a triangular fragment in the middle third of the leg on July 16, 1951. On July 27, 1951, osteosynthesis was performed with a vitallium Lane plate in anatomic position with 2 to 3 mm. diastasis. A walking plaster cast was applied in the after-treatment. Structural callus formation was observed on January 29, 1952.

CASE II. (Table 1, No. 24, Fig. 7.) A fifty year old man was admitted with a compound transverse fracture in the middle of the leg. The transverse wound measured 6 cm. On September 11, 1948, within six hours of the accident, a Lane osteosynthesis was performed with two plates, in anatomic position with a diastasis of 3 mm. A walking plaster was used in the after-treatment. An Unna dressing was applied on January 25, 1949. Structural callus formation was observed on March 8, 1949.

CASE III. (Table 1, No. 30.) A nineteen year old man was admitted with a transverse fracture in the middle of the leg. A Lane osteosynthesis was performed on September 14, 1949, three days after the accident. The position was anatomic with a diastasis of 2 mm. The fixation with a V_2A steel plate was inadequate, leading to a 10° valgus position in the plaster cast. The plaster was removed on January 17, 1950, and the patient went to work on February 13, 1950. Structural callus formation was found at a subsequent check-up on January 12, 1951.

CASE IV. A thirty year old man was admitted with an oblique fracture in the middle third of the leg. On March 26, 1951, two days after the accident, osteosynthesis with a vitallium Lane plate was performed in anatomic position with a 2 mm. diastasis. A walking plaster was applied in the after-treatment. An Unna dressing, applied on June 19, 1951, was probably too early. A second trauma on September 6, 1951, led to bending of the plate and refracture. Active treatment was not given for this new fracture, only a walking plaster being used. Structural callus formation was observed on January 22, 1952.

These four cases show that diastasis should be avoided in osteosynthesis. On the other hand, if diastasis is present, it does not necessarily lead to pseudarthrosis, provided there is adequate fixation for a sufficiently long period.

A second important factor in delayed callus formation is the severity of the trauma. Our series contains two cases with major trauma. From the very first day we must be prepared for the healing in such cases to take a long time.

Osteitis constitutes the third factor. Structural callus formation cannot be expected so long as there is osteitis. If the fixation permits, all foreign bodies (including the vitallium) must be removed in such a case.

Atrophy is the fourth factor. If the bone is atrophic, we must also expect slow callus formation, and it is certainly wise to combat this atrophy by means of general treatment and activation of the function.

Pseudarthrosis

Table 1 lists six cases with pseudarthrosis. In five of them mistakes were made in the treatment; if they had been discovered in time, pseudarthrosis might have been prevented.

CASE I. (Table 1, No. 26.) A twenty-seven year old man was admitted with a complicated open double fracture of the leg. The perforating wound showed lacerated muscles and there were also multiple abrasions of the skin. The whole impression was of a sack with loose bones. On May 28, 1949, the day of the accident, osteosynthesis with three Lane plates was performed after wound excision. To close the wound over the osteosynthesis, the skin was mobilized by means of a relief incision. Free split-skin grafts were used to close this secondary incision. A plaster cast was applied in the after-treatment. Healing of the wound was uneventful. On June 28, 1949, a walking plaster

was applied above the knee, and on October 18, 1949, below the knee.

The mistake in the after-treatment was made at this stage, for starting from this moment, the fixation was inadequate. We should exert more patience before proceeding to consolidation after such a severe trauma.

The proximal Lane plate proved to be broken on December 13, 1949. The patient did not wish to have a new operation. On February 6, 1950, the distal fracture was consolidated. The proximal end was still unconsolidated. On February 10, 1950, the patient was treated in another hospital with an onlay graft taken from the tibia of the other leg. Structural consolidation was found on June 22, 1950.

Case II. (Table 1, No. 79.) A twenty-six year old man was admitted with a complicated open fracture. It was major trauma caused by a tree falling on his leg; he had a perforating wound with lacerated skin and muscles. On March 30, 1951, the day of the accident, osteosynthesis with a vitallium Lane plate was performed after wound excision. The skin defect was closed with pinch grafts on April 25, 1951. The plaster was removed on May 11, 1951. Here again the mistake was made in the after-treatment, leading to insufficient fixation. We should have waited longer before removal of the plaster. Real pseudarthrosis was observed on December 17, 1951. It was removed and an inlay graft from the other tibia was applied, fixated with vitallium screws. On May 17, 1952, the patient showed constructive consolidation and normal function.

Case III. (Table 1, No. 43b.) A thirty-four year old man had had a bad motorcycle accident which had caused a complicated transverse fracture of the proximal third of the tibia and fibula. The patient had undergone plaster treatment elsewhere, but severe pains made him consult us on July 8, 1949. We found that the fracture was in bad position with a lateral and longitudinal displacement. The skin was infected. This was treated with the leg in wire extension. Osteosynthesis was performed on July 20, 1949, followed by after-treatment with plaster. The knee was put in plaster on November 8, 1949. The after-treatment proved to be inadequate, fixation not being sufficient. Constructive consolidation did not develop.

The patient consented to reoperation on June 2, 1950. Plate and screws were removed and a Beck's drilling carried out through the pseudarthrosis. After-treatment was with plaster. Consolidation was observed on September 5, 1950.

Case IV. (Table 1, No. 34.) A twenty-seven year old man had an accident on June 12, 1949, which caused a transverse fracture in the middle third of the leg. Osteosynthesis was performed on June 13, 1949; after-treatment was with plaster which was removed on August 23, 1949. This was too early because the fixation subsequently proved to be inadequate. The x-ray showed absence of constructive callus formation. The callus was resorbed between the fragments. On October 19, 1949, the plate showed a fracture. It was removed with the screws and a Beck's drilling performed through the pseudarthrosis with after-treatment in plaster. Consolidation was observed on February 7, 1950.

Case V. (Table 1, No. 83.) A twenty-eight year old man had a motorcycle accident on June 1, 1947, which caused a complicated oblique fracture with a large wound in the middle third of the leg. Treatment was by wire extension and plaster after six weeks. On January 16, 1948, we found bad callus formation and a bad position. Osteosynthesis, although late, was resorted to. The reduction was not anatomic. This was a mistake, but provided the fixation in plaster was good, we could leave it at that. On February 10, 1948, a plaster was applied distal to the knee. This was the second mistake, for from then on the fixation was insufficient. A fracture of the plate and of one screw was observed on May 8, 1948. Between the fragments the callus was resorbed. Pseudarthrosis operation (Kirschner) was performed on July 16, 1948. Plate and screws were removed, and the bone chiseled away over a distance of 2 inches.

The sclerotic fragments were replaced by cancellous chips from the ilium. After-treatment was with plaster, in good position without internal fixation. Structural callus formation was observed on February 22, 1949. The patient was re-examined on February 26, 1952. The leg had both normal form and function, and there was no disability. This case shows that the main cause of pseudarthrosis formation is not the patient's constitution, but the severity of the trauma, bad reduction, insufficient fixation and infection. If these factors are borne in mind, pseudarthrosis may be prevented in

many cases; at most slow callus formation may occur in some cases.

CASE VI. (Table 1, No. 47, Fig. 9.) This was also discussed as Case 1 under "Osteitis." We know that pseudarthrosis will develop, and that treatment of it must be postponed until the soft tissues are in good condition, with good mobility and good circulation. The osteitis must have subsided and the bone must show evidence of reconstruction. At present, with the aid of modern antibiotic treatment, this waiting period can be much shorter than it was in this instance.

If pseudarthrosis had developed, grafting, a Kirschner operation and drilling according to the Beck method would have given good results.

Disability

One hundred fourteen patients showed structural callus formation after about four months, and the period of disability was between four and six months. In the case of slow callus formation the longest duration is eight and a half months (No. 36); in pseudarthrosis, four years (No. 47, Fig. 9). All patients have recovered with good function.

CONCLUSIONS

1. The use of the Lane plate should be considered in osteosynthesis of the tibia.
2. Vitallium completely comes up to the requirements of an inert material.
3. Indications and technic are of great importance. Mistakes are always reflected in the callus formation.
4. Osteosynthesis with the Lane plate requires after-treatment without full weight bearing.
5. The drawback of the diastasis in the use of the Lane plate is not important, provided the fixation period is sufficiently long. The picture of diastasis is often only a matter of organization and is not real.
6. If we are dealing with open fractures, osteosynthesis should only be performed on narrow indications. This operation is not allowed under war conditions at the front.
7. Lane plate and screws must be removed if osteitis has developed. Further treatment is possible with plaster, provided the position is good.
8. Operative treatment of pseudarthrosis should be postponed until the osteitis has subsided.

Resuscitation after Fifty Minutes of Cardiac Arrest

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WHEN the heart has lost its ability to propel blood, either by standstill or ventricular fibrillation, cardiac arrest exists. Fautex[1] defined true cardiac resuscitation as "full and durable restoration in due time to avoid eventual death, of the essential functions of a heart which has lost its power to propel blood and which, in all appearances, is unable to recover spontaneously its effective value as a pump." He also stated the following to be criteria of true cardiac resuscitation: "The heart must have ceased functioning as a pump and cardiac function must have been fully and permanently restored and life preserved."

We have gained insight into the rapid effects of cerebral anoxia in animal experimental work. It has been reported that when the pulmonary artery of a cat was clamped in forty seconds breathing ceased. Spontaneous recovery occurred if the clamp was removed within three minutes; between three and four minutes resuscitation was necessary, and after four minutes permanent neurologic changes or death occurred. In the human being, according to Mousel et al.,[2] complete cerebral anoxia for ten seconds produces unconsciousness; for twenty to thirty seconds it causes cessation of electroencephalographic brain waves; for three to five minutes it produces irreversible pathologic changes in the cerebrum. The medulla and its centers can survive for twenty-five to thirty-five minutes, and the spinal centers from forty-five to sixty minutes. Several investigators have found changes in the cerebral cortex characterized by individual cell degeneration, a patchy area of necrosis, diffuse zonal necrosis and complete cortical degeneration.

Even though life may be preserved if anoxia advances to the point where irreversible brain damage occurs, there will be permanent clinical manifestations. The individual may be a vegetative organism. Sequelae which have been reported are deafness, blindness, athetoid and choreiform movements, Parkinsonism, emotional instability and speech defects. Vernon[3] recorded a case in which there was prolonged recovery from subdiaphragmatic massage after an arrest of four minutes. The patient regained consciousness the morning after the massage. For two weeks he spoke a few incoherent phrases; then he talked nonsense incessantly. After a month he became maniacal, requiring hospitalization in an observation ward. He remained there for five months and then went to a convalescent home. At nine months he had a violent tremor of the hands, slurring of speech and often repeated words. At eleven months he was well enough to start work.

As has been established then by experimental investigation and clinical experience, the diagnosis must be made and treatment instituted in cardiac arrest within a period of four minutes if we are to preserve life.

Incidence. From the 1,200 cases collected by Stephenson et al.[4] we learn that cardiac arrest is a much more frequent occurrence than is generally realized. As the authors state, there are considerable data indicating that the actual incidence of cardiac arrest is increasing although this has yet to be definitely confirmed. One is surprised in learning from their collected cases that of all those reported, 21 per cent were patients under ten years of age. These include arrest encountered during operations upon patients with congenital heart disease, but these cases do not constitute unduly significant members in the series. After this peak the age incidence fell and rose gradually as age increased up to seventy years; the age incidence then dropped sharply.

Certainly the surgeon is more aware of this complication and is recognizing it more often. It is reasonable to expect that cardiac arrest will occur more often in the future than in the past because of the rapid development of surgery of the thoracic viscera.

Johnson and Kirby[5] reported four cases in two years at the hospital of the University of Pennsylvania. Hinchey and Straehley[6] reported four cases within a six-month period in a 250 bed hospital. Cooley[7] reported an incidence of 5.5 per cent, or once in every eighteen cases, in patients being operated upon for pulmonary stenosis at the Johns Hopkins Hospital. There were forty-eight cases in 878 operations. Miller et al.[8] reported fourteen cardiac arrest fatalities in 12,000 surgical cases at the University of Minnesota Hospitals. This is a ratio of one in every 858 cases done under general anesthesia. Estimates have been made that the incidence is somewhere between one case in every 3,000 to 5,000 operations. However, as cases are reported, the indications are that the incidence is higher than we have believed in the past.

Etiology. In discussing the etiology of cardiac arrest, one must realize that in most cases the true cause is not known. As one reads the literature and listens to discussions concerning this condition, he is struck with relationship of anesthesia and the anesthetic agents used, and such factors as hypoxia, hypercapnia and vagal stimulation. Basically, the two factors involved are anoxia and vagal stimulation. Anoxia may vary in degree and may be due to any one of several causes. Inadequate preoperative blood level and inadequate blood replacement during surgery, whether the blood loss be due to sudden severe hemorrhage or gradual continued loss, contribute to anoxia. As far as anesthesia is concerned, factors producing anoxia are inadequate airway producing complete occlusion or prolonged hypoxia, excessive deep plane of anesthesia and overmedication before or during surgery.

There is dispute concerning the role of reflex stimulation of the vagus nerve. Many authorities believe that vagal stimulation by its inhibitory action on the heart beat predisposes to cardiac arrest. Stephenson et al.[4] emphasize the importance of the relationship of the vagovagal reflex and the fact that anoxia and hypoxia potentiate this reflex.

Sloan[9] has shown in laboratory animals that vagal stimulation is not productive of cardiac arrest in the absence of hypoxia or hypercapnia. However, there have been repeated reports of cardiac arrhythmia or arrest coincident with the insertion as well as withdrawal of an endotracheal tube. In the series of Stephenson et al.[4] reliable data indicating a precipitating factor prior to arrest were obtained in 624 cases. Of the 156 cases in which vagovagal reflex action seemed likely, eighty-five were associated with endotracheal intubation, bronchoscopy or esophagoscopy, twenty-five were associated with pulmonary hilar trauma, and fourteen at the time of direct contact or cutting of the vagus nerve.

When one is operating near the hilus of the lung, carotid sinus or vagus nerve, he must be aware of the possible vagovagus effect. Even though there has been no apparent cause demonstrated in a number of cases of cardiac arrest, one must be aware of hypoxia, anoxia and hypercapnia as well as the vagal stimulating effect as etiologic factors.

DIAGNOSIS

When the surgeon is operating within the thoracic cavity, he may be the one who first detects cardiac arrest; but even then it may be the anesthetist who is first aware of asystole. It is the anesthetist who is first aware of cardiac irregularity, hypotension and respiratory distress before there is an actual cardiac arrest. After he has notified the surgeon of these difficulties or the inability to obtain peripheral arterial pulsation and blood pressure, little time is wasted in trying to establish a positive diagnosis. If the operation is an intrathoracic procedure, the heart is easily visualized or palpated. In an abdominal procedure one can palpate the aorta or common iliac arteries. If neither the abdomen nor thorax is opened, one may insert a needle into the heart and observe its hilt for movement produced with heart beat. The needle should be at least 3 inches long. If a suitable needle is not immediately available, valuable time should not be lost in getting one.

It has been emphasized that various diagnostic procedures, including visualization of the pulsations of the retinal vessels with an ophthalmoscope, inhaling amyl nitrite to stimulate forceful contractions in a feebly beating heart, observing the speed of capillary refill and examination of an electrocardiographic tracing, are either unreliable or impossible. If the heart beat is too feeble to cause peripheral arterial pulsation or to sustain the blood pressure, it is not likely to be heard with a stethoscope. Valuable time is lost in conjecture when one debates the possibility of a

cardiac standstill versus ventricular fibrillation or a feebly beating heart. The heart has ceased to function as a pump regardless of the state it is in and the decision calls for prompt intelligent action. Since failure in successful resuscitation is due chiefly to delay between arrest and cardiac massage, no valuable time is lost; the thorax is opened by the easiest and quickest method. This is the only conclusive diagnostic procedure. If one is operating in the abdomen, the diaphragm may be divided to gain access to the heart; otherwise the approach is transthoracic. In the latter case, sterility is of secondary importance. All one needs is a knife and gloves. A quick incision between the fourth and fifth ribs anteriorly will determine if there are cardiac pulsations. The incision can be enlarged by dividing the adjacent costal cartilages in order to massage the heart.

PROPHYLAXIS

Just as diagnosis and treatment of cardiac arrest require cooperative effort between the anesthetist and surgeon, so does the prophylaxis. As has been repeatedly stressed by those writing on this subject, most cases of cardiac arrest are preventable. In the preoperative preparation of the patient attention must be given to bringing him into surgery in the best state of health to reduce the possibility of cardiac arrest. Attention is given to fluid and electrolyte balance, correction of anemia, regulation of cardiac drugs when indicated and the choice of preoperative medication. Atropine in therapeutic doses should be used to help prevent vagovagal stimulation.

During the operation the most important of all prophylactic measures is the anesthetic management of the patient. Mousel et al.[2] state that the intelligent management of the patient in the preoperative period and during anesthesia will prevent most of the harmful sequelae. The maintenance of adequate oxygenation is of extreme importance. Ruth,[10] reporting for the Anesthesia Study Commission, stated that the most common error during the period preceding cardiac arrest was that the anesthetist tolerated anoxia in his patient. Ruth also advised minimizing trauma and manipulation while the patient is in a light plane of anesthesia because these tend to set off dangerous reflexes. The proper selection and skillful administration of anesthetic agents is also of great importance. In the series of Stephenson et al.[4] almost one-half of the patients had either two or three anesthetic agents, and a sizable percentage had four. There have been differences of opinion concerning the use of procaine intravenously or topically to the pericardium during surgery in the thorax when it is known that the heart will be traumatized. There is agreement among all who concern themselves with the problem of cardiac arrest that most cases are preventable when attention is given to the foregoing prophylactic measures.

TREATMENT

Just as the diagnosis and prophylaxis of cardiac arrest require cooperation between the anesthetist and surgeon, so does the treatment. Treatment resolves itself into the main purposes of early and adequate oxygenation of tissue, especially the brain, and production of adequate blood flow by cardiac massage until normal heart beat is restored. Adequate oxygenation depends upon adequate ventilation of the lungs by artificial methods until respiratory function is restored. Whether or not life is preserved and brain damage is prevented depends upon these facts.

Ideally, the urgent need for oxygen is best met with an endotracheal tube and 100 per cent oxygen administered by the anesthetist. Artificial respiration by normal compression of the breathing bag 16 to 20 times per minute is the most satisfactory method. If an endotracheal tube is not in place, valuable time should not be lost in introducing one unless it can be done with rapidity. After the patient is well oxygenated, using a well fitting face mask, an endotracheal tube may be inserted. One should make certain there is no obstruction to the air passages if thoracic excursion is inadequate. However, exploration of the oropharynx and insertion of an airway should not hinder unduly the administration of oxygen.

In the cases of Stephenson et al.[4] 87 per cent of the cardiac arrests occurred in the operating room. If they occur outside the operating room, while one is securing anesthetic equipment exhaled air by the mouth-to-mouth technic may be used. To produce pulmonary ventilation, there are various types of resuscitators; Kreiselman's produces an intermittent flow of oxygen under adjustable positive pressure. Others are the alternative positive and negative pressure types which cause rhythmic inflation of the lungs followed by suction. In spite of their

advantages they produce damage to the lungs and a harmful effect to the circulation; also, the efficiency is greatly reduced when they are not used properly. Mousel et al.[2] state that these resuscitators should not be relied upon in such emergencies. Since 87 per cent occur in the operating room and a significant number of the remaining 13 per cent occur during induction of anesthesia, anesthetic machines are available. Their use in pulmonary ventilation is preferable to mechanical resuscitators.

In the establishment of cardiac rhythm the most important therapeutic aid is cardiac massage by the surgeon's hand. As previously stated, one is not primarily concerned with sterility; the emphasis is on cardiac massage without undue delay. In Stephenson's study the most common denominator in the unsuccessful cases was that of delay. The thoracic approach is preferable to massage through the diaphragm and today it has almost supplanted the abdominal or subdiaphragmatic route. By the thoracic approach, one actually visualizes the heart and can arrive at a more accurate diagnosis. Ventricular fibrillation can be diagnosed better and the heart is more accessible for electrical defibrillation. The heart can be more efficiently massaged and the aorta can be occluded below the origin of the vessels of the arch in order to force as much blood as possible to the brain and coronary arteries. When the patient is put in the Trendelenburg position, it does not interfere with massage.

There is difference of opinion as to how fast the heart should be massaged. Hinchey and Straehley[6] are among those who suggest a rate of 40 to 60 per minute. It has been suggested that a slower rate be maintained in order to allow the ventricles to fill adequately. Johnson and Kirby[5] studied the efficiency of heart massage in dogs. They found, in comparing the rate of massage of 30, 60 and 120 per minute, that a greater blood flow was produced when the heart was compressed at a rapid rate. They advised, in clinical use, the establishing of a rate as rapidly as possible up to 120 times per minute. However, as they bring out, one cannot continue this rate of massage for more than a few minutes. A rate of 80 per minute could be maintained if the surgeon and his assistant alternate. In the same study the authors found the technic of placing the thumb in front and the fingers behind to be the most effective method of compression in the dog's heart and advised this method for a small human heart. They also found that only about one-fifth as much blood flow was produced by compressing the heart through the diaphragm as with the method described heretofore. They advised placing one hand in front and one hand behind the heart in compressing the usual adult heart.

One question looms before anyone who has experienced the necessity to massage the heart for arrest and there is no restoration of heart beat after several minutes: When should one cease to massage the heart? In Stephenson's series the heart was restarted in 56 per cent of the 1,200 cases. The heart started to beat after an arrest of over forty-five minutes in one case reported by Beck et al.[11] The patient recovered with no signs or symptoms of central nervous system injury. Other cases of arrest of forty minutes, twenty-seven minutes and twenty-five minutes with resuscitation have been reported. In one of our cases there was no perceptible heart action for fifty minutes. The heart was massaged for sixty minutes and the patient resuscitated with no signs or symptoms of central nervous system injury.

Stephenson et al.[4] state that "As long as massage appears adequate, a good peripheral pulse is maintained and the pupils remain contracted, efforts should be continued."

In view of the reported cases, one concludes that prolonged and persistent effort is certainly worth while.

Drugs. All authorities are agreed that medication is no substitute for cardiac massage when arrest occurs. However, several drugs have been suggested to aid in bringing about a more forceful beat as well as reducing myocardial irritability.

It has been suggested that 1:1000 epinephrine solution be injected directly into the heart if it does not begin to beat within a few minutes after normal massage has been started. There is debate as to the strength of the solution and the site of injection. Some are hesitant about injection into the left ventricle because the concentrated epinephrine might have rapid access to the coronary circulation. It has been suggested that injection into the auricle is less likely to cause ventricular fibrillation.

Epinephrine produces a more forceful contraction by a direct effect on the myocardium and on the conduction tissue.[2] The objections raised to its use are: (1) It increases myocardial

irritability. (2) It elevates the oxygen consumption of the heart muscle. (3) It increases the auriculoventricular conduction time. Consequently, it has been suggested that procaine hydrochloride in 1 per cent solution be administered at the same time epinephrine is given. Lahey[12] advised a combination of 4.75 cc. of 1 per cent procaine and 0.25 cc. of 1:1000 epinephrine injection into the left ventricle. Procaine hydrochloride diminishes the irritability of the conduction system of the heart and of the myocardium. However, Fautex,[1] as well as others, believes that procaine does not prevent fibrillation. Toxic effects have been reported by Read[13] and Denney et al.[14] The effects were ventricular acceleration and fibrillation which resulted in death in one instance.

Barium chloride will help strengthen the heart beat. The injection of 1 or 2 cc. of 0.5 per cent solution is a safe procedure and will support the heart after contractions have been restarted by massage. Atropine sulfate in doses of $\frac{1}{50}$ and $\frac{1}{100}$ gr. is a valuable drug when one suspects a vagovagal reflex. It may be administered intravenously.

If the cardiac arrest is a result of exsanguination, there is no substitute for whole blood. Veal et al.[15] have shown that there is an immediate rise in the systemic arterial pressure and a concomitant rise in the coronary artery pressure when the transfusion is given by the intra-arterial method. Therefore this is preferable over the intravenous route. If there is profound collapse and one cannot use a peripheral artery, time should not be lost in such efforts. The aorta is available and may be used for intra-aortic transfusion. The venous return to the heart may be increased after spontaneous contractions have been initiated by applying Esmarch's bandages from the toes to the groin of each leg.

Ventricular Fibrillation. If the heart ceases to function as an efficient pump due to ventricular fibrillation, treatment assumes different aspects. Beck and Mautz[16] advise cardiac massage as the initial treatment. If rhythm is not established in a period of one or two minutes, they then advise injecting 5 cc. of 1 per cent procaine into the right ventricle and 5 cc. into the pericardium. Meanwhile, the massage is continued. The procaine elevates the threshold of cardiac muscle to incoming stimuli and blocks out stimuli which may be causing and continuing fibrillation. If massage and procaine are ineffective, electrical shock is indicated. This is accomplished by placing electrodes on each side of the heart and passing alternating currents of 60 cycles, 1.0 to 1.5 amperes and 110 volts through it for a very brief period, about one-half second. This passage of electrical current through the heart will cause simultaneous contractions of all the fibers which have been contracting arrhythmically. Complete relaxation of the fibers results and the fibrillation changes to cardiac standstill. Then normal spontaneous rhythm may be resumed after a brief period of standstill or it may be necessary to massage the heart to initiate contractions. If it is necessary to repeat the procedure, intravenous atropine is given. Before electrical shock is instituted, it is important that anoxia be overcome by cardiac massage and artificial ventilation of the lungs with 100 per cent oxygen. Beck and Mautz[16] have demonstrated the importance of the strength of the current used. In animals, 0.4 amperes for five seconds caused fibrillation while 0.8 amperes or more stopped it. Also, a current of 0.8 amperes will not cause fibrillation while 0.45 amperes will not stop it. Herrod et al.[17] demonstrated on animal hearts that repetitive stimuli will not revive the heart in a state of standstill due to anoxia but are effective in standstill due to vagal stimulation. Their investigation showed that rhythmic manual compression is more effective than repetitive electrical stimuli in reviving a heart in standstill.

In Stephenson's cases[4] there were 133 in ventricular fibrillation, of which eighty-four were successfully defibrillated. Thirty-nine patients permanently survived. Therefore one need not consider ventricular fibrillation as a hopeless state. Approximately one patient of ten was in a state of ventricular fibrillation and one-third of these were resuscitated.

An apparently simple, safe and effective electrical device for defibrillation that is also quite inexpensive has been described by Hinchey and Straehley.[6] They credit this device to Buckley, Koewn and Ruth who, with Baily at Hahnemann Hospital in Philadelphia, have used it effectively. This device consists of two electric cords, 150 cm. long, leading from an ordinary wall plug to two copper electrodes 8 by 5 cm. in width and about 2 mm. thick. Buckley et al. suggest that one or more electrical shocks be administered

to the fibrillating heart by placing a copper electrode on each side of the heart and then making connection from the attached plug to the ordinary wall current of 110 volts and 60 cycles of alternating current. The current is immediately disconnected after the surgeon counts "one, two, three." The entire body of the patient may jerk convulsively. If necessary, the shock may be repeated once or several times. The authors believe that the degree of electrical shock to the heart is effective and the danger is negligible. They suggest insulating the electrodes that are applied to the heart to protect the surgeon's hand, and placing the connection to the plug and the wall current a safe distance from the operating table to minimize the hazard of anesthetic explosion.

Shepard[18] describes an improved cardiac defibrillator which is apparently a safe and effective device that is commercially available. It is also simple to operate from an ordinary wall plug.

CASE REPORTS

CASE I. A. C., a forty-two year old white woman, was admitted to Mercy Hospital with a history of recurrent pulmonary infections involving the left lung. Physical examination revealed decreased resonance and reduced to absent breath sounds in the left upper thorax. Roentgenologic study revealed atelectasis of the left upper lobe of the lung. Bronchoscopic examination was made and no lesion was visualized. Cytologic study of the secretions aspirated was not conclusive for malignant cells. After preoperative preparation, using pentothal,® nitrous oxide, oxygen, demerol® and curare as the anesthetic agents, left thoracotomy was performed.

At operation a large tumor mass was found originating in the left upper lobe. There were no visible or palpable nodes in the hilar region but there were multiple secondary tumor masses in the upper lobe. Our decision was to perform pneumonectomy because we were of the opinion that lobectomy would be too conservative for this lesion. After ligation of the pulmonary artery the lung was elevated in order to visualize better the posterior hilar region. At this time deep cyanosis suddenly developed. The anesthetist was unable to produce an exchange of the anesthetic gases by positive pressure through the endotracheal tube. The heart ceased to beat and the cyanosis deepened. Cardiac massage at the rate of 60 strokes per minute was instituted by the surgeon while the anesthetist attempted to clear the air passage. The bronchoscopic equipment was prepared when the anesthetist was successful in aspirating, through the endotracheal tube, a piece of tumor tissue. This allowed a free airway. With the administration of 100 per cent oxygen and continued cardiac massage, in about four minutes there was a perceptible heart beat. There were occasional extrasystoles for a period of about two minutes and then a regular rhythm was established. The cyanosis cleared rapidly after the air passage was open. Gross examination of the piece of tumor revealed it to be a cast of a large bronchus.

The lung was removed with no further difficulty. The lesion proved to be an undifferentiated small cell carcinoma. One hour after surgery the patient responded well. There were no signs or symptoms of central nervous system damage when she was dismissed from the hospital on the eleventh postoperative day. She is now gainfully employed as a stenographer.

CASE II. T. R., a forty-seven year old white man, was admitted to Mercy Hospital because of recurring right pulmonary infection, cough, sputum and a weight loss of 23 pounds. Physical examination revealed this man to be chronically ill with evidence of weight loss and findings in the right upper thorax in keeping with atelectasis. Roentgenologic study revealed an infiltration with partial atelectasis of the right upper lobe. Bronchoscopic examination revealed a tumor mass in the region of the orifice of the right upper lobe. The specimen removed for study proved to be carcinoma.

After preoperative preparation using nitrous oxide, oxygen and ether as an endotracheal anesthetic, right thoracotomy was performed. The carcinoma was very extensive. It involved all three lobes of the lung and was attached to the pericardium. There were large hilar lymph nodes extending along the trachea. Since this man was having a febrile course with obstruction to the bronchus, we elected to perform palliative pneumonectomy. On two occasions we observed mild cyanosis during the exploration of the extent of the lesion. This was discussed with the anesthetist who was having difficulty keeping the patient well oxygenated.

While the tumor was being dissected away from the pericardium, the heart ceased to beat. Cardiac massage was instituted immediately. Five cc. of 2 per cent procaine were applied to

the pericardium. Pulmonary ventilation was accomplished through the endotracheal tube using 100 per cent oxygen. After five minutes the heart beat was restored with regular rhythm. We chose not to proceed with the palliative pneumonectomy and the thorax was closed. The patient never regained consciousness postoperatively. He was comatose, did not respond to painful stimuli and died forty-eight hours after surgery.

CASE III. C. F., a forty-eight year old white man, was admitted to Wesley Hospital because of a cough productive of bloody sputum, pleuritic pain and weight loss. There were reduced breath sounds in the left lower lobe. At bronchoscopic examination a lesion was visualized in the left lower lobe bronchus. A biopsy proved this to be undifferentiated carcinoma.

After preoperative preparation left thoracotomy was performed using pentothal, nitrous oxide, oxygen, demerol and curare as the anesthetic agents. A large carcinoma originating in the left lower lobe was found. Enlarged lymph nodes were palpable in the hilar region. It was our decision to perform pneumonectomy with hilar dissection. Three ligatures were placed around the pulmonary artery which was then divided between the two distal ligatures. There was an immediate massive hemorrhage. At that time our technic did not include placing a hemostat on a portion of the arterial wall before it was severed. However, we were successful in grasping the opened pulmonary artery between the thumb and index finger. It was difficult to estimate the blood loss accurately. The heart ceased to beat and no pulse or blood pressure was obtainable. A hemostat was placed across the artery and cardiac massage was started while the anesthetist administered 100 per cent oxygen through the endotracheal tube. Since an 18 gauge needle was present in an arm vein and a 15 gauge needle in a leg vein, transfusions of whole blood were given under pressure. The heart was collapsed and flabby due to the lack of blood volume. It did not readily refill after it was emptied by compression. After 2,000 cc. of blood had been given, it began to refill palpably after compression. A rate of 60 compressions per minute was established using alternate hands with the thumb on one side and the four fingers on the opposite side. The pericardium was not opened. After thirty-five minutes of massage and artificial pulmonary ventilation with 100 per cent oxygen we contemplated the advisability of continuing the resuscitation measures. However, the pupils were not widely dilated and there seemed to be capillary filling although there was no perceptible peripheral pulse or blood pressure. We were of the opinion that he would probably have irreparable brain damage. However, we continued the resuscitative measures; and fifty minutes after it had ceased to beat, the heart had the first perceptible beat. The beat was feeble and irregular, and therefore massage was continued for ten minutes more. The heart was then regular in rhythm, the pulse was discernible and the blood pressure returned to the preoperative level. The cyanosis cleared and spontaneous respiration was established. During the sixty minutes the patient received continuous cardiac compression, 100 per cent oxygen was administered by the anesthetist and 5,500 cc. of whole blood were given. No procaine, epinephrine or other drugs were used.

While the patient continued to receive 100 per cent oxygen, the lung was removed. He responded during the stimulation of the skin closure. When he was removed from the operating room, he spoke incoherently. He was confused as to time and place for five days. Electrocardiographic study was interpreted as showing diffuse myocardial ischemia nine days after surgery. The patient was dismissed from the hospital to his home eighteen days after surgery. One month postoperatively he had no evidence of central nervous system impairment. Three months after surgery a neuropsychiatrist examined him. His wife, who was mentally ill, gave a history that necessitated neuropsychiatric consultation for both of them. He was found to have no signs or symptoms pertaining to the central nervous system and had the stability of personality to withstand the psychic trauma of the death of his wife with metastatic brain cancer. He died eight months after pneumonectomy from metastasis but had no sequelae from the cardiac arrest.

This case strikingly illustrates the success possible in resuscitating a heart in arrest when proper measures are instituted without delay. It also bears out the advice to continue resuscitative measures as long as they seem adequate and the patient's pupils remain contracted.

In the first case, arrest was brought on by complete occlusion of the trachea with resulting anoxia; the second case is illustrative of hypoxia

and dissection in the region of the pulmonary hilus, while the third case was due to massive hemorrhage with exsanguination.

COMMENT

It is evident that cardiac arrest is amenable to prompt and definite therapeutic measures. There are also data indicating an actual increase in the incidence. Therefore it behooves every busy surgical group to set up a program of action to meet this emergency. A sterile cardiac emergency kit should be kept in each operating room, anesthesia room, emergency room and x-ray department. This is borne out by the study of Stephenson et al.[4] which showed that arrest is most likely to occur while the patient is in these places. The kits need not be elaborate but, as emphasized by Lahey,[12] they should contain two of everything because of the excitement and confusion resulting in such emergencies. The most important components of the kit are instruments to open the chest, two scalpels, two scissors, two tissue forceps, two hemostats and two pairs of gloves. The sufficient drugs are two 5 cc. ampuls of 1 per cent procaine, two 1 cc. ampuls of 1:1000 epinephrine, two ampuls of atropine and two ampuls of 0.5 per cent barium chloride or calcium chloride. Two 5 cc. syringes, two 18 gauge needles and two 22 gauge needles will complete the kit.

A cardiac defibrillator should be present in each surgical suite. The copper electrodes of the defibrillator should be kept sterile. The entire unit should be mobile and accessible.

This subject should be presented to the hospital staff annually with instruction as to the availability of the kits and the importance of prompt, intelligent action.

SUMMARY

In cardiac arrest the diagnosis must be made and proper treatment instituted within four minutes if resuscitation is to be successful. The basic factors in producing cardiac arrest are anoxia and vagal stimulation. Since delay is the main factor contributing to failure in resuscitation, a program of management with a cardiac arrest kit should be set up in every busy surgical group. Prophylaxis will reduce the incidence, and proper management will resuscitate a greater percentage of patients. Cardiac massage by the thoracic approach and pulmonary ventilation using 100 per cent oxygen are the effective means to prevent serious brain damage. The use of drugs and the administration of whole blood as indicated are valuable adjuncts in therapy. Ventricular fibrillation has been successfully treated, and simple safe effective defibrillators are available. Three illustrative cases have been reported, one being a successful resuscitation after fifty minutes of cardiac arrest. A program of action should be set up in every hospital to meet this emergency.

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In Vivo Experiments with Carcinogens

A SURVEY FROM 1952 TO 1953

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THIS study cites some 240 references to *in vivo* experiments with carcinogens for the period from 1952 to 1953 which have not been covered in previous surveys.[173]

INORGANIC CHEMICALS

Radioiodine. Large doses of I-131 produced pituitary tumors in mice.[60,64,65] Silberberg[191] found that intraperitoneal injections of the isotope in young mice caused more hypophyseal tumors than in older animals. Several investigators warn of the induction of thyroid cancer by the administration of I-131.[41,63,212]

Radiogold. Williams[229] noted a high incidence of hepatomas in rats given Au-198 by the splenic route and then fed 3'-methyl-4-dimethyl-aminoazobenzene.

Radiophosphorus. Moore et al.[133] used P-32 to produce adenocarcinoma in the stomach of mice.

Uranium. Hueper et al.[91] induced sarcoma in the bone and chest walls of rats by the use of uranium. Smith[199] cited the occupational hazards of uranium miners in Germany and Bohemia.

Nickel. Smith[199] also noted the hazards in the nickel refineries of Great Britain, and Hueper[92] described sarcoma in 30 per cent of rats injected with nickel powder.

Chromates. Two studies warn of the respiratory neoplasms in workers in the chromate industry.[20,202]

Arsenic. Arsenical cancers from exposure to medical and occupational arsenic have been studied.[201]

Cobalt and Zinc. Thomas and Thiery[215] produced liposarcoma in rabbits with cobalt or zinc.

Lead. Zollinger[242] caused papillomas and carcinomas in rats with lead phosphate.

Bentonite. Wilson[231] described the production of hepatomas in eleven of twelve mice fed bentonite, a base exchange silicate.

Asbestos. Boemke[16] noted the formation of lung carcinoma from the inhalation of asbestos.

Zinc Chloride. Injections of $ZnCl_2$ in the testes of chickens resulted in carcinoma that led to teratoma.[26]

Phosphate. Na_3PO_4 increased the growth of Ehrlich carcinoma and benzpyrene-induced tumors.[213]

HYDROCARBONS

Methylcholanthrene. This remains the most used carcinogen of the hydrocarbons. Deringer et al.[35,83] induced pulmonary tumors in mice and guinea pigs by giving methylcholanthrene intravenously. Thamsen[214] produced tumors in some strains of mice by homografts of lung tissue with the hydrocarbon. Gastric neoplasms resulted in mice and rats from intramural injections of the hydrocarbon.[79,174,204] Hitchcock[90] noted squamous cell carcinoma of the stomach in mice given the carcinogen by stomach tube. Croton oil has been used to enhance the carcinogenicity of methylcholanthrene in mice.[98,112-114] Estrogens and methylcholanthrene showed a synergistic action in the production of tumors in mice.[74,107] The number of epidermal methylcholanthrene-induced tumors in mice was increased by subcutaneous injections of cortisone.[208] The hydrocarbon induced carcinoma and fibroma in ducks.[158,160,161] Russell et al.[168,169] caused different types of sarcoma in guinea pigs by subcutaneous injections. Shay et al.[186–188] studied the influence of sex hormones on tumor production in methylcholanthrene-treated rats, and also the transfer of Myelogenous leukemia induced in rats. Saxén[178] induced more sarcomas in younger than older mice with the hydrocarbon. Rogers and Rous[166] investigated the joint action of methylcholanthrene and a neoplastic virus to induce carcinoma in rabbits. Skin carcinogenesis by the carcinogen has been studied further by Cambel,[25] Marchant and Orr,[126] and Wolbach.[232] Laurens and Bacon[118] induced sarcoma in the wall of the rectum of a rat using methylcholanthrene. Carcinogenesis with the hydrocarbon has been investigated under various

types of stress.[116,155,233] Strong[206] reported further observations on survival time of mice bearing methylcholanthrene fibrosarcomas.

Benzpyrene. Two studies[239,240] have been made on the production of benzpyrene tumors in hypophysectomized rats, and two investigations[29,147] have been carried out on the carcinogenicity of metabolite products of the hydrocarbon. The atmospheric pollution by benzpyrene has been considered.[219] Graffi et al.[67] noted the carcinogenic activity of croton oil and benzpyrene. Engel,[50] Schober,[182] and Wolbach[232] contributed further to the carcinogenicity of the hydrocarbon.

Dimethylbenzanthracene. Mosinger[135] induced sarcoma in guinea pigs with single implantation of this hydrocarbon. Saxen[176] studied squamous cell carcinoma of the forestomach in x-irradiated mice fed dimethylbenzanthracene. Ritchie et al.[162,189] and Salaman[171] increased tumor production in mice by repeated use of croton oil after application of dimethylbenzanthracene. Ritchie[164] also investigated the effect of arterial occlusion on dimethylbenzanthracene carcinogenesis in the rabbit. Geyer et al.[61] noted the synergistic effect of estrogens and dimethylbenzanthracene in carcinogenesis in rats, while Gwynn and Salaman[73] described the co-carcinogenesis of dimethylbenzanthracene and SH-reactors in mice. Linell and Norden[121] contributed further to dimethylbenzanthracene carcinogenesis in rabbits. Iversen[97] studied early effects of the hydrocarbon in mice.

Dibenzanthracene. This has been used to induce lung tumors in mice.[33,34,87]

Saxen and Stewart[177] produced gastric sarcoma in mice by injections of the hydrocarbon. Shubik et al.[190] produced skin carcinomas by painting mice with the hydrocarbon. A single application of dibenzanthracene followed by croton oil treatment caused skin hyperplasia in mice.[165]

Benzanthracene. Steiner and Edgecomb[203] produced sarcomas in mice by subcutaneous injection of this. Iversen et al.[98] studied the effect of croton oil on benzanthracene carcinogenesis in mice.

Petroleum. Eby et al.[48] discussed the occupational hazards from the carcinogenicity of petroleum products. Tests on the carcinogenicity of petroleum products in mice and rats were conducted by Hieger and Woodhause,[89] Hueper,[95] and Schmähl and Reiter.[180] Paraffin wax pellets produced papillomatoses in rats.[18]

Tar. Smith[199] has evaluated the risks of occupational lung cancer in workers exposed to coal tar fumes. Fisher[57] noted folliculitis, chronic erythema, etc., in tar workers in England.

Phenanthrenes. Methyl homologs of 1,2-cyclopentenephenanthrenes injected into mice produced carcinoma.[23]

Chrysenes. 3-Methyl chrysene applied to mice was weakly carcinogenic.[66]

ORGANIC NITROGEN COMPOUNDS

Azo Compounds. Feeding rodents dimethylaminoazobenzene (DAB) induced hepatomas.[51,80,81,108] Nelson and Woodard[145] noted carcinomas in the liver, urinary bladder and gallbladder of dogs fed DAB. Graffi et al.[67] included DAB in their study of croton oil carcinogenesis. Haranghy et al.[78] fed guinea pigs cholesterol and DAB, and injected benzpyrene to develop sarcoma and lymphosarcoma. Miller et al.[129] reported that fluoro derivatives of DAB were more carcinogenic to rats than DAB. Druckrey[42] investigated the long latent period of DAB tumors.

3′-Methyl-4-dimethylaminoazobenzene (3′-methyl DAB) produced cancer of the liver[10,222] and splenic tumors.[10] Williams[229] fed rats 3′-methyl DAB and administered Au-198 by the splenic route to produce a high incidence of hepatomas. Graffii et al.[67] used this azo compound with croton oil in their study of carcinogenesis in mice. Price et al.,[152,153] and Miller and Miller[127] employed 3′-methyl DAB and 4′-fluoro DAB in their investigations on liver tumors in rats. Druckrey[42] demonstrated the long latent period of DAB tumors. MacDonald et al.[123] demonstrated the synergistic action of 3′-methyl DAB and other azo compounds.

Corre-Hurst et al.[30] found that meta and para derivatives of butter yellow showed additive effects but that 4-hydroxyazobenzene showed a negative effect in the production of cancer of the liver. Mulay et al.[55,56,140–143] studied liver tumors produced by p-dimethylaminobenzene-1-azo-1-naphthalene. Badger and Lewis[4] concluded that the most active azo compounds were amino, methylamino and dimethylamino derivatives of azobenzene for producing hepatomas in rodents. Sugiura

et al.[207] reported that only one ethyl derivative of aminoazobenzene was carcinogenic. Ray and Peters[156] coupled m-cresol with diazotized 2-aminofluorene to produce gastric lesions in rats. o-Aminoazotoluene induced carcinomas in the liver, gallbladder and urinary bladder of dogs[145] and the same carcinogen caused hepatomas in mice.[1]

Fluorenes. 2-Acetlyaminofluorene (AAF) produced tumors of the bladder,[134,195] liver,[12,123,128,134] breast[13,128,167,179] and thyroid.[3,217] Mosonyi[136] described the rapid production of hepatomas in rats fed AAF and given tannic acid subcutaneously. MacDonald et al.[123] noted the synergistic action of AAF and 3′-methyl DAB in production of tumor of the liver. Dyer and Morris[47] caused tumors in rats by gastric intubation with AAF, aminofluorene and 2-nitrofluorene. Cambel and Ray[24] induced papillomas and gastritis in rats with AAF derivatives, while Bielschowsky[11] gave n-monomethyl- and n-dimethyl-2-aminofluorene by stomach tube to induce hepatomas.

Aromatic Amines. Walpole et al.[221] caused a number of neoplasms in rats by subcutaneous injections of 4-aminodiphenyl and 3,2′-dimethyl-4-diphenyl. Sandin et al.[174] caused mammary tumors in rats with acetylaminodiphenyl. Beta-naphthylamines caused neoplasms in mice and dogs.[17,184] Baker[5] produced adenocarcinoma of the colon and sarcoma of the stomach in rats fed dihydroxybenzidine.

Urethane. Urethane has been used widely to induce lung cancer in mice.[6,22,71,109,110,125] Croton oil increased its activity.[67,172]

Thiouracils. Thiouracil,[131] methylthiouracil[41] and propylthiouracil[132,185,217,218] have been employed to produce thyroid neoplasms in rodents. Propylthiouracil also induced pituitary neoplasms in rodents.[132,185]

Nitrogen Mustards. Narpozzi[144] produced skin cancers in mice with HN2. Sarcomas have been noted in mice given nitrogen mustard and podophyllin.[46] Heston et al.[85,88] induced pulmonary tumors in mice with HN2.

Aminostilbene. Elson[49] caused carcinoma of the lung, liver, and other carcinoma in rats on a high protein diet with several stilbenes.

Dibenzcarbazole. Hepatomas have been noted in mice following subcutaneous injections with 3,4,5,6-dibenzcarbazole.[1]

Pyrodixine. This agent increased the number of takes of transplanted sarcomas in rats.[53]

Sulfonamides. Hansen and Bichel[77] noted 20 to 25 per cent occurrence of tumors in rodents given sulfonamides.

Indole. Kaiser[99] was unable to produce tumors in rats given indole for 590 days.

Amino Acids. Popper et al.[150] produced hepatomas in rats fed ethionine. Ritchie[163] noted more papillomas in mice given adrenalin subcutaneously.

HORMONES

Estrogens. Heston et al.[84] found 20 per cent mammary tumors in mice given pellets of cholesterol plus 10 per cent diethylstilbestrol. Huseby[96] caused mammary and pituitary cancers in castrated mice on a diet which included the estrogen. Diethylstilbestrol or alpha-estradiol given with dibenzanthracene produced more mammary tumors than the hydrocarbon alone.[61] Dunning et al.[43] employed estrone pellets to induce adenocarcinoma and squamous cell cancers in rats. Williams et al.[230] concluded that carcinoma of the cervix was produced by alpha-estradiol, but Bayly et al.[9] discredited the influence of estrogens in stimulating this type of neoplasm. Hall et al.[74] noted the synergistic action of estrogens and methylcholanthrene in the production of tumors of the uterus in mice, and Kirschbaum et al.[107] described similar results in their study of leukemogenic agents. Kirkman and Bacon[106] induced kidney tumors in golden hamsters with diethylstilbestrol or estradiol.

Gonadotropin. This hormone promoted the successful takes of transplants into the spleens of castrated rats.[243]

Cortisone. Sulzberger et al.[208] increased the number of methylcholanthrene-induced epidermal tumors in mice by using cortisone. Brownell et al.[21] and Foley[58] noted that this hormone and desoxycorticosterone also promoted the growth of lymphosarcoma and adenocarcinoma in mice. Mirand et al.[130] caused fibrosarcoma and myosarcoma in mice given the latter hormone. Smith[197] described larger tumors in rabbits which were given cortisone from the appearance of tumors by fibroma virus. Cortisone also increased the growth of tumors in rodents and fowls.[69] Werder et al.[225,226] made mice more susceptible to leukemia by cortisone treatment.

Adrenal and Anterior Hypophysis Transplants. Silberberg et al.[192] noted that both transplants increased the incidence of leukemia in young, castrated mice.

MISCELLANEOUS

Smoking. The relation of smoking to lung cancer has called forth a number of investigations.*

Croton Oil. The promoting action of croton oil in carcinogenesis has already been noted.[67, 98, 111, 113, 114, 165, 171, 189] Ritchie et al.[162] used repeated applications of croton oil following one application of a hydrocarbon in his study of carcinogenesis. Peacock et al.[148] included croton oil in their study of dietary carcinogenesis.

Mammary Tumor Agent. The mammary tumor agent has been studied extensively.[2, 7, 14, 15, 82, 137–139, 149, 151, 154]

Cell and Tissue Suspensions and Extracts. Injections of cell and tissue suspensions and extracts have been employed to induce neoplasms.†

Viruses. Smith,[197] and Kilham and Woke[105] induced neoplasms in rabbits with fibroma virus. Rogers and Rous[166] inoculated rabbits with the Shope virus and a chemical carcinogen to produce carcinomas.

Transplants. Law[119] caused leukemia in mice with thymic extracts. Snell et al.[200] studied the growth of S-37 and S-180 transplants in mice of various strains.

Enzymes. Luhrs and Willig[122] described the accelerating effect of a hyaluronidase on the growth of Jensen's sarcoma in rats. Tedeschi[213] reported that cocarboxylase increased the growth of carcinoma and sarcoma.

Occupational. Attention has been directed to the occupational hazards in the uranium and nickel industries.[199] Two reports called attention to the carcinogenicity of isopropyl alcohol production.[199, 224] Risks to tar workers[57] and gas workers[37] have also been investigated. Precautions in other industries have been considered.[28, 93–95]

Dyes. Certain dyes fed to rats caused lymphosarcoma.[227, 228]

Trypan Blue. Trypan blue induced sarcomas in mice[62] and rats.[194] Kaliss and Borges[101] noted the synergistic effect of tumor tissue and trypan blue in tumor production.

Podophyllin. This agent induced sarcomas in mice.[46]

Cholesterol. Heston et al.[84] described mammary cancer in mice caused by pellets of cholesterol and estrogens. Cholesterol and carcinogenic hydrocarbons induced sarcoma in guinea pigs.[78]

Iodoacetic Acid. Repeated application of this chemical following a single application of a carcinogen caused a number of neoplasms.[73]

Mustard Gas. Vapor of mustard gas induced lung tumors in mice.[86]

Alkaloids. Tumors of the liver developed in rats fed Senecio alkaloids.[183]

p-Benzoquinone. Application of this chemical to mice induced papillomas but no carcinomas.[216]

Plastics. Plastics embedded in rodents caused fibrosarcoma, sarcoma and other neoplasms.[146]

Barnacles. These placed subcutaneously in mice produced sarcomas.[241]

Diet. Heavily smoked bacon caused tumors in rats.[181] Semi-purified diets enhanced tumor production in the liver of mice.[193] Moderate amounts of vitamin B_{12} in the diet of mice led to faster tumor growth than low or high B_{12} diets.[210] Critical decrease in dietary protein increased mammary carcinoma in mice.[211] Mice made obese by gold thioglucose showed earlier mammary carcinoma than controls.[223] Flexner-Jobling carcinoma[120] grew as fast in starved rats as in well fed rats.

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The Heredity Factor in Cancer of the Breast

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THE cause of cancer is still unknown even though many strides are being made and more is being learned about the disease every day. Many factors have been considered as causative agents and some have been found to play definite roles in the etiology. Among these is the heredity factor which, although it probably is not the sole cause, must be considered a predisposing cause in certain families. As Slye[4] concluded, inheritance of cancer susceptibility alone is probably not always sufficient to insure the occurrence of cancer, yet cancer probably results from external factors acting with internal factors on susceptible soil.

A number of authors have recorded clinical evidence of the importance of heredity as a causative factor in the formation of cancer. Macklin[3] reported on fifty-three pairs of monozygous twins. Of these, thirty-two pairs both showed tumors, and thirty-one of thirty-two tumors were of the same type. Vandenberg[7] cites a family in which a mother and two daughters had cancer of the left and then of the right breast, and a granddaughter by the first daughter had cancer of the left breast. There was also a family history of "cancer" in the mother's mother and two brothers. He also cites Warthin's Family G in which a patient died of gastrointestinal cancer. In the next generation six of ten relatives over twenty-five years of age died of cancer, and in the third generation twenty-seven of sixty-four relatives over twenty-five years of age had cancer. The tumors in the family were almost completely confined to the gastrointestinal tract. In addition, Vandenberg[7] reported the case of a woman seen by him with a blue-black mole on the forearm. Her sister and mother had similar lesions and in the identical location. Tendler[5] reported cancer of the breast in three sisters and in a maternal aunt and great-aunt.

Little[2] cites Bittner's experimental work on mice: He crossed females of a high breast tumor strain with males of a low tumor strain, and obtained an incidence of breast tumor in the female offspring of over 90 per cent. On crossing females of a low tumor strain with males of a high strain the incidence of tumor in the female offspring was less than 5 per cent. Since the chromosomal complex in the offspring of the two groups was identical, he believed that some powerful influence other than chromosomes was active. He therefore crossed two more groups in the same way but fostered the female offspring to other mice for nursing. The resulting tumor incidence was as follows:

	Nursed by Females from High Strain	Nursed by Females from Low Strain
Hybrid female from high strain female and low strain male	95.0% tumors	1.3% tumors
Hybrid female from low strain female and high strain male	88.6% tumors	1.4% tumors

These findings would indicate that breast milk is an important factor in the origin of cancer of the breast. This is no doubt true, but in our group of cases this could not be a factor since the familial susceptibility apparently came from the father rather than the mother.

What then is the factor that caused cancer of the breast to develop in these women? It would certainly seem that it is more than just a matter of chance. Trout[6] suggests that there may be an abnormality of hormonal function in women with cancer of the breast: "In pelvic conditions associated with painful breasts during the menstrual periods, sometimes a simple dilatation of the cervix, but frequently more extensive pelvic surgery, is indicated, and such will relieve both the pelvic condition and the breast pain. It is suggestive to note that over 60 per cent of our cases of carcinoma of the breast gave a history of association of pain in the breast just previous to the menstrual period, whereas in a similar series, without carcinoma of the breast, only about 20 per cent had painful breasts associated with menses.

"Often Theelin, prolactin or some similar injection started three to four days before menstruation will prevent painful breasts during that period. A few cases require the addition of antuitrin-S, some are benefited by thyroid extract, and an occasional patient requires epinephrine."

Lacassagne[1] is more specific about the response of the breast to hormonal stimulation in these families. He has shown that in susceptible strains of mice weekly injections of estrone provoke cellular proliferation in the epithelial cells of the breast and soon induce cancer, while in the strains not normally susceptible no cancer results. He suggests that the breast in certain families may have a tendency to retain estrogen more than normal and it may, upon becoming stagnant, become carcinogenic or be transformed into a carcinogenic agent.

In our hospital we have treated two sisters and a half-sister with cancer of the breast. Their case histories are as follows:

CASE REPORTS

CASE I. Miss A. L. L., a twenty-six year old white woman, was admitted on February 4, 1952, with a non-tender mass filling the upper, inner quadrant of the left breast of two weeks' known duration. There had been no such previous trouble. The mass was not attached to skin or deep tissues and did not transilluminate. No axillary nodes were felt. On February 5, 1952, the lesion was excised and found to be cancer, therefore radical mastectomy with dermatome skin graft from the left thigh was performed immediately. The pathologic report revealed the lesion to be adenocarcinoma with axillary metastases.

On February 12, 1952, bilateral oophorectomy was performed with the hope that this would help slow the growth of any metastases that might be present. A course of postoperative deep x-ray therapy was completed on April 14, 1952.

In January, 1953, the patient suffered a broken back in an accident but there was no evidence of pathologic fracture. When she was examined on June 23, 1954, there was slight edema of the left arm but motion was excellent. The patient remained in good health until June, 1955, when she was admitted through the accident room with acute pain in the back following a fall. X-rays revealed metastasis to the seventh dorsal vertebra. No sign of any other recurrence could be found at this time.

CASE II. Mrs. L. L. J., a nineteen year old white woman, was admitted on February 8, 1952, with a small lump in the lower portion of the right breast and another beneath the upper outer portion of the left nipple. Although her half-sister (Case I) had been admitted to the hospital several days before with a similar complaint, the patient denied that that was the reason she came in for a check-up.

On February 9, 1952, both lumps were excised and found to be benign pericanalicular fibroadenomas. After the wounds healed, the patient was examined carefully every six months. There was some persistent fibrosis beneath the scar but this did not feel suspicious until September, 1953. Three weeks before this the patient noted a firm, non-tender lump in the left axilla. She did not believe this had enlarged since she first noticed it. Examination at this time revealed induration in the region of the previously noted fibrosis without attachment to the skin but with apparent attachment to the underlying tissue. On September 12th the lesion was excised and found to be malignant; radical mastectomy was therefore performed immediately. On September 21st bilateral salpingo-oophorectomy and appendectomy were performed. Some of the skin of the wound sloughed, so on September 29th a dermatome graft was applied. A course of deep x-ray therapy was given following healing of the wound. The patient was last seen in January, 1955, in good health with no evidence of recurrence. She was reported to be well in every respect in June, 1955.

CASE III. Mrs. D. L. M., a thirty-one year old white woman, was admitted on February 19, 1952, with a hard mass in the far lateral side of the right breast without attachment to the skin but with seeming attachment to surrounding breast tissue. The patient came in for a check-up only because her sister (Case I) had been found about a week before to have cancer of the breast, and her half-sister (Case II) had been found to have lumps in her breasts. The patient denied any breast trouble except for soreness before menses. When the mass was found on examination and called to the patient's attention, she said that she had forgotten about it as it had been there since an injury to the region seven years previously.

On February 20, 1952, the mass was excised and found to be malignant. Radical mastectomy was performed immediately, and on February 27th bilateral salpingo-oophorectomy and appendectomy were performed. The pathologist reported the lesion to be adenocarcinoma and no sign of malignancy could be found. She appeared to be in excellent general condition and was feeling fine. The dyspnea had disappeared.

In June, 1955, although the patient was in good general health, a mass developed in the

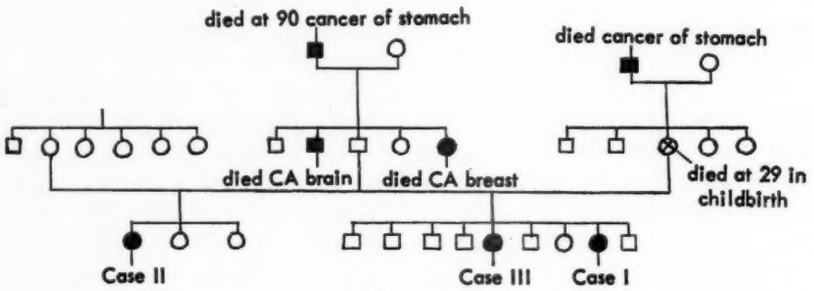

FIG. 1.

with axillary metastasis. A course of postoperative deep x-ray therapy was given.

The patient's hospital course was uneventful. She got along well until July, 1952, when pneumonia of the right lower lobe developed which did not respond to treatment at home. She was readmitted to the hospital from August 1 to September 13, 1952, and treated for pneumonia. A metastatic lesion of the chest was considered. The white blood count was normal throughout the course. The pneumonia improved on treatment and the patient was discharged and followed up as an outpatient.

At a visit on September 30, 1953, the patient revealed that she had a great deal of pain throughout the chest; but this was about the same as it had been since the original operation two years previously. Examination of the left breast, right chest wall, axillas, skull, spine, pelvis, and neck revealed no nodules or tenderness.

Examination on April 2, 1954, revealed more shortness of breath than there had been a few months previously, and the patient felt run down. Examination revealed no nodules but there was tenderness just above the right eye in the frontal regions. X-rays revealed opacity in the right frontal sinus region, but serial x-rays were read as not being indicative of metastasis. The lung condition was not clear to x-ray but was improved over previous ones and not thought to be due to metastasis.

On June 23, 1954, the patient was examined left axilla, the opposite side from the original tumor. A hard gland was removed and found to be metastatic spread of the same type as the original tumor.

The history of cancer in this family is shown in Figure 1. This is interesting in that the only parent common to all three patients is the father, and it is on his side of the family that most of the other cases of cancer occurred.

SUMMARY

Heredity as a factor in the causation of cancer of the breast has been discussed. Case histories of cancer of the breast in three young women, two sisters and a half-sister, have been added to the literature.

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The Efficacy of Diaparene Chloride on the Sterility of Wet Surgical Linen

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OWENS,[6] and Colebrook and Hood[1] have demonstrated the transmission of bacteria through moist dressings. Similarly Propst[7] reported upon the contamination of the operative field by the passage of bacteria through wet surgical linen. Furthermore, in this study it was shown that the addition of roccal,* a quaternary ammonium compound, to the final rinse of the surgical linen resulted in a marked decrease in the incidence of contamination. The study was limited to the fall and winter months, and all cultures were taken in air-conditioned operating rooms in which the mean temperature was 72°F.

Recently the authors had the opportunity to observe the efficacy of another quaternary ammonium compound, diaparene chloride,† in the prevention of contamination of wet surgical linen. This work was undertaken during the summer months in operating rooms which were not air-conditioned. The temperature in these rooms varied from a low of 78°F. to a high of 98°F., with a mean temperature of 82°F. The results of this study are presented herein.

MATERIAL AND METHOD

Diaparene chloride is an odorless, colorless, stable, cationic germicide, the chemical composition of which is di-isobutyl cresoxy ethoxy ethyl dimethyl benzyl ammonium chloride, monohydrate. In studies on mice, guinea pigs, rabbits, dogs and chicks it has been found to have a low order of acute and chronic toxicity. Diaparene chloride is a proved bactericidal agent for Escherichia coli, Staphylococcus aureus, Streptococcus viridans, Streptococcus faecalis, Pseudomonas pyocyanea, Aerobacter aerogenes, Proteus vulgaris, Streptococcus hemolyticus and Alcaligines faecalis.[5] The clinical material for this study was obtained during the performance of forty-seven major operations. This series included procedures in both the abdomen and the thorax. The majority of operations were upon the gastrointestinal tract.

In one study the caps and shirts used by the surgical team were immersed for ten minutes at 45°C. (113°F.) in an aqueous solution of diaparene chloride (1:5000) and compatible synergists. (This time and temperature were chosen as approximating that of the final rinse used by most laundries.) The volume of the solution was varied to make available different quantities of diaparene chloride for exhaustion on the fabric. These quantities ranged from 1 to 2 ounces of active ingredient per 100 pounds of cloth. In another, a control study, the caps and shirts used were not impregnated with diaparene chloride. Patches of linen 2 inches square were inserted into pockets on front of the "scrub" shirt and in the fold of the "scrub" cap. (Fig. 1.) The caps and shirts, both impregnated and unimpregnated, were then subjected to the standard sterilization technic. The room temperature and the duration of each operation were recorded. At the termination of the operation and under aseptic precautions all patches were transferred to a 5 per cent solution of tamol N, a quaternary inactivator. The solutions were immediately implanted on blood agar, proteose No. 3 agar, and MacConkey agar plates. At twenty-four- and forty-eight-hour intervals total and differential bacteria colony counts were made.

RESULTS

A total of sixty-two caps were used. Thirty-eight were impregnated with diaparene chloride and twenty-four unimpregnated caps were used as a control. When the temperature was less

* Product of Winthrop-Stearns, Inc., New York, N. Y.

† Product of Homemakers' Products Corp., New York, N. Y.

FIG. 1.

than 86°F., no patch, either in an impregnated or in an unimpregnated cap was contaminated. This temperature was the lowest at which soiling of the cap with perspiration was observed. When the temperature was 86°F. or higher, contamination occurred in one of twenty-seven (3.7 per cent) impregnated caps and in nine of nine (100 per cent) control caps. In operations in which the room temperature (86°F. or higher) and the duration of the operation were comparable the incidence of contamination in the impregnated caps was one of nine (11.1 per cent), and in the control study it was nine of nine (100 per cent).

In a study of the incidence of contamination derived from the moisture of the surgeon's skin combined with the moisture of the operative field, the "scrub" shirt and the contained patches were used. The patches in the shirt were separated from the operative field in front by two layers of linen, namely, the front of the "scrub" shirt and the surgeon's sterile gown. In back the patches were separated from the surgeon's skin by one layer of linen, the "scrub" shirt. In one series forty-seven shirts were impregnated with diaparene chloride and forty-seven unimpregnated shirts were used as a control study. Contamination of the patches in the unimpregnated shirts occurred at the lowest recorded temperature, 78°F. The incidence of contamination in the impregnated shirts was five of forty-seven (10.6 per cent), and in the unimpregnated shirts it was twenty of forty-seven (42.5 per cent). The shirts treated with diaparene chloride appeared to resist contamination for a longer period of time than the untreated shirts.

The bacterial strains isolated from the contaminated cultures were Staphylococcus albus, hemolytic and non-hemolytic staphylococcus aureus and Str. hemolyticus. A comparative study of the bacterial colony counts in the contaminated impregnated shirts and the contaminated unimpregnated (control) shirts was made. In the contaminated impregnated shirts the average colony count per culture of the non-hemolytic staphylococcus aureus and hemolytic staphylococcus aureus was 51.5 and 20, respectively. In the contaminated unimpregnated shirts the average colony count per culture of non-hemolytic and hemolytic staphylococcus aureus was 962.3 and 60, respectively.

COMMENT

In clean postoperative wounds Staph. aureus or albus has been reported as a dominant organism in 62 per cent of the wound infections that occurred.[3] These bacteria are permanent residents in the hair follicles and sebaceous glands of the normal skin and are not removed by the usual mechanical and chemical cleansing of the operative field.[2] During operation, when the skin is moistened by perspiration or wound secretions, these organisms become a potential source of wound contamination. Staphylococci grow best in an environment having an alkaline pH of 7.8 and during the summer months when the skin is constantly moist. Diaparene chloride is most effective when used in an alkaline medium.[5] However, in the presence of serum and organic matter diaparene chloride and other quaternary ammonium compounds function as less efficient germicides.

In the present study, when contamination of wet drapes by hemolytic staphylococcus aureus did occur, the bacteria colony count was three times greater in the control linen than in the linen impregnated with diaparene chloride. When it is recalled that approximately 30 per cent of the hemolytic staphylococcus aureus are resistant to penicillin,[4] both the relative and

absolute reduction in contamination by the staphylococci subsequent to the use of diaparene chloride are believed of practical importance.

In this experiment diaparene chloride in a concentration of 1:5000 was used. Its effectiveness in this concentration was demonstrated by the presence of contamination in only five of forty-seven (10.6 per cent) impregnated shirts. This is believed significant when compared to the presence of contamination in twenty of forty-seven (42.5 per cent) of the unimpregnated or control shirts. Furthermore, neither toxic nor allergic reactions to the use of diaparene chloride (1:5000) were observed.

CONCLUSION

Diaparene chloride (1:5000) is believed to be an effective germicide in the prevention of contamination of wet surgical linen.

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Pulmonary Embolism and Infarction

DIAGNOSIS AND MANAGEMENT

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PULMONARY embolism is very frequently overlooked or misdiagnosed in clinical practice. Surgeons are apt to confuse it with postoperative atelectasis (Case I) until active thrombophlebitis becomes evident. General practitioners are prone to confuse it with virus pneumonia until the patient calls attention to the pain in his leg. We have treated two physicians, each of whom had been so diagnosed by their confreres. One had "four attacks" before the phlebitis in his saphenous vein was given its proper significance. Patients with rheumatic heart disease have been treated for multiple episodes of pneumonia over a period of years until embolization finally has been correctly diagnosed, as in the instance of Case II. On careful study patients diagnosed as having lung disease proved to have suffered from a myocardial infarction with pulmonary embolism from a mural thrombus.

Farmer and Smithwick[1] have reported on 7,343 consecutive surgical admissions. In ninety-five of these pulmonary infarction was recognized; in twenty-five pulmonary infarction was the first indication that phlebitis was present.

The terms pulmonary embolism and pulmonary infarction are not synonymous. A pulmonary embolism means that a foreign body, most commonly a portion of a thrombus, has lodged in a branch of a pulmonary artery with or without changes in the lung tissue supplied by that branch. If there is a profound change in the lung tissue which progresses to necrosis, the term infarction is used. Emboli in the absence of infarction do not usually produce detectable x-ray changes unless opaque visualization studies are performed. If the disturbed area is near the lung surface, however, changes in physical signs will be noted, especially the finding of rales or a pleural friction rub. The work of Kjellberg[5] aids in understanding this phenomenon. He subjected a series of dogs to artificial pulmonary embolization. No roentgenographic changes in the lungs were observed in uncomplicated pulmonary embolism, and no infarction occurred as long as the circulation through the bronchial artery was intact. Infarction usually extends to the surface of the lung. The irritated pleura reacts by an outpouring of fluid. Pleural effusion is thus a frequent finding. Blood exudes through damaged capillary walls into alveolar spaces. Therefore hemoptysis is a common finding.

SYMPTOMS

Pain is often the first and most compelling symptom. Two types of pain are encountered: (1) that produced by reaction of the pleura overlying the infarction and, (2) severe anterior chest pain often indistinguishable from that of myocardial anoxia. It sometimes produces "splinting" and difficulty in breathing. Some workers believe that this pain is the result of an actual diminution in the coronary flow secondary to the profound disturbances in the local hemodynamics.[2,3,4] Others hold that this is due to pain in the pulmonary artery itself. This question has not been settled as yet.

Dyspnea is common. It is due to the splinting caused by pain.

Cough with hemoptysis of slight or marked degree is a most important corroborative sign. Without it a diagnosis is very difficult to maintain. We have seen patients, however, with convincing evidence of pulmonary embolization without hemoptysis.

The emotional reaction may be one of panic and fear of disaster. This depends on the patient's whole personality, but the panic reaction is sufficiently common to warrant comment.

SIGNS

Fever will depend on the degree of infarction or secondary pneumonitis, and the presence or absence of phlebitis at the site of origin. Some fever is usually present and it may be marked.

Heart rate and rhythm: Tachycardia is present in proportion to the fever. Arrhythmias are often present and are responsible for many emboli, especially in rheumatic heart disease. Any patient who is fibrillating is a candidate for pulmonary emboli. We have seen a patient in normal rhythm have an episode of paroxysmal fibrillation, discharge an embolus and revert to normal rhythm in a period of several hours. It is our impression that it is the recently formed thrombus or recently propagated new clot superimposed upon the old clot in a heart or vein that embolizes.

Signs of consolidation, dullness to percussion, bronchial breathing rales and a pleural friction rub may be present if the infarct is large enough. Over areas of pleural effusion diminished breath and voice sounds will be heard. As the effusion increases, flatness develops. Careful examination should at least show rales somewhere in the chest to support the diagnosis.

FINDINGS

Laboratory: Leukocytosis with an increase in polymorphonuclear leukocytes is usually present depending on the presence or absence of phlebitis and pneumonitis. The sedimentation rate increases rapidly during the first week.

X-ray: The presence of a shadow in the lung fields will only be found if some degree of infarction has occurred. The shape, size and density of the shadow depends on the angle which the infarct makes to the direction of the x-rays. Thus, shadows vary from small, hazy, mottled areas to large, dense segments. The classic pie-shaped area, while sometimes seen, is not found in the majority of cases. Some degree of pleural effusion is usually seen in the x-ray films sometime during the course of the illness.

Electrocardiographic: A large pulmonary embolus, by blocking a generous percentage of pulmonary artery flow, produces an increase in pulmonary artery pressure. This in turn is reflected in the heart by dilation of the right ventricle and what is termed "right heart strain." The electrical changes are a shift of the axis to the right with clockwise rotation. This gives the following changes in the leads:

I Deep S, depressed ST
III—Q, elevated ST
aVR—R, elevated ST
aVL—depressed ST, inverted T
aVF—elevated ST
V_1 and V_2—R prominent, elevated St VAT prolonged
V_4, V_5, V_6—ST depressed, S marked in V_5, V_6

Variations from this pattern depend on the size and location of the embolus.

COMPLICATIONS

Cor pulmonale, acute dilation of the right ventricle and pulmonary conus may follow massive pulmonary emboli. These changes may produce signs which can be recognized on physical examination. Distention and increased pulsations of the veins of the neck are common signs. Increase in pulsation may also be noted in the second and third interspaces to the left of the sternum. The second pulmonary sound may be heard over the area of increased pulsation. Gallop rhythm heard best to the left of the sternum may be present.

Shock: "Peripheral circulatory collapse" may be present and require emergency measures. While we recognize that shock is a variable manifestation, most patients we have encountered in shock from pulmonary embolization display cold, moist, pale hands; moist face; cyanosis; weak, feeble, rapid pulse and hypotension.

DIFFERENTIAL DIAGNOSIS

Space does not permit consideration of all conditions which must be considered in a differential diagnosis. The diagnosis will be made more often if it is kept in mind. All patients with cardiac arrhythmias, rheumatic heart disease, recent myocardial infarcts and phlebitis are good candidates.

PREVENTION AND TREATMENT

In a sense pulmonary embolism may be likened to lightning. It strikes most often with great suddenness; and if the patient survives the initial shock, he has a good chance of surviving that particular embolus. It is the emboli yet to be delivered that are most to be feared; indeed, many large fatal pulmonary emboli are preceded by small ones. As Barker[6] pointed out, if all fatal pulmonary emboli preceded by minor emboli could be prevented, the death rate would be strikingly reduced.

Autopsy statistics in large series show a rate of 10 per cent pulmonary embolism in this country.

For this particular syndrome the best treatment is definitely prevention. A review of a large number of case histories has shown that a greater awareness or a more meticulous study on the part of the physician would have alerted him to signs which might have resulted in the initiation of prophylactic measures. For example, charts of patients who died from pulmonary embolism have contained notes such as, "patient complains of pain in left calf" or "pain in the right groin" or "sudden breathlessness" or a "sharp pain in the right chest." Many other examples could be cited. Because these initial signals were of short duration, disappearing within a few days or even hours, their sinister significance was not recognized and steps were not taken to prevent further development of thromboembolic complications. As Barnes pointed out fifteen years ago, little could be done except wishful thinking if the pulmonary emboli were arising from the heart. If they were arising from the veins of the legs, ligation proximal to the site of a recognized thrombophlebitis appeared logical and was the favored method of attack. Experience has demonstrated, however, that (1) ligation does not prevent the active thrombophlebitic process from continuing; (2) emboli may and frequently do continue to arise from veins other than those ligated; (3) fatal emboli may arise from thrombi which have formed immediately proximal to the site of ligation, even though this may be as high as the inferior vena cava; (4) the late effects of ligation of the femoral, iliac or inferior vena cava veins are often undesirable with serious evidence of venous insufficiency with pain, edema and ulceration. Therefore, ligation was found not to be a preventive measure of choice except when it was desirable to eliminate varicose veins, a common site for recurrent thrombophlebitis with secondary emboli.

Erb and Schumann[7] reported their experience in 100 cases of fracture of the femur, a condition very frequently complicated by pulmonary embolism. Fifty patients were subjected to bilateral femoral vein ligation and fifty were used as controls. The mortality rate was not lowered in the surgical group; indeed, the ligation itself was frequently followed by thrombosis in the site proximal to the ligature. Pulmonary embolism was increased.

That surgical interruption of veins is not a treatment for phlebitis is well illustrated by a patient of ours. While in the Army he had a sudden pulmonary embolus. Examination disclosed phlebitis in his left leg. Left superficial femoral vein ligation was performed. In the convalescence from this operation a second pulmonary embolus developed. Examination disclosed phlebitis in his right leg. This time, in an effort to trap all possible emboli, his inferior vena cava was ligated. Subsequently phlebitis developed in his arms and he again had a pulmonary embolus. Since his physician did not wish to tie off the superior vena cava, he was referred to us for long-term anticoagulant therapy. There have been no further emboli during a period of six years.

The advent of anticoagulant drugs permitted a new approach to the problem, which is actually based on the fact that under certain conditions the blood clots too easily and pieces of the original clot break off and travel to the lungs. If the original clot could be prevented from forming, no such problem would ever arise. If such a clot, once formed, could be contained as a small process and allowed to seal itself off instead of propagating a tail which could break loose, the risk would be markedly lessened. If emboli once delivered could be prevented from propagating and blocking off more branches of the pulmonary arterial tree, the risk to life would be decreased. All of these accomplishments are theoretically possible with the perfect anticoagulant used correctly under favorable conditions. All of them have been achieved in varying degree, and often with striking success, with the presently available anticoagulants. Many studies have demonstrated a striking reduction in mortality and morbidity when anticoagulants have been used either prophylactically prior to the first pulmonary embolus or even after the first embolus in the prevention of subsequent ones.

A summary of the results of several large series of patients with postoperative thrombophlebitis shows the following. Once thrombophlebitis has been recognized in the absence of anticoagulant therapy, the risk of pulmonary embolism ranges from 4 to 60 per cent. Of those patients who have one embolus, the risk of death from a subsequent embolus is

approximately 20 per cent. In contrast, patients with postoperative thrombophlebitis who receive adequate anticoagulant therapy suffer from pulmonary emboli in less than 5 per cent, and the mortality rates are under 0.5 per cent.[8] These are indeed striking figures. They have been duplicated with heparin, heparin and the coumarin derivatives combined, and the coumarin derivatives alone. In obstetric cases[9] the incidence of pulmonary embolism following untreated thrombophlebitis has been reported as ranging from 15 to 35 per cent with a mortality rate of 3 to 5 per cent. This low mortality may be associated with the finding of the Committee on Anticoagulants[10] that while the younger patients had nearly as many emboli as older patients, the mortality rate was much lower, probably because of their ability to stand the reaction or shock. In addition, it is probable that more of the pulmonary emboli in obstetric patients arise from pelvic veins of smaller caliber and hence are less likely to be fatal.

While it appears that once a thrombus or a pulmonary embolus has been recognized anticoagulant therapy is the most important form of therapy, certain other measures should be borne in mind.

When carrying on a physical examination of the chest the physician should scrupulously avoid having the patient inhale deeply or cough since such maneuvers increase the negative venous pressure, thus developing a most favorable condition to suck loose the tail of a clot which may then travel into the lungs. Deaths have occurred directly following such maneuvers. The patient must be warned against straining at stool, thus producing a Valsalva experiment following which the hemodynamic pressures are markedly disturbed. In one series 20 per cent of the deaths from pulmonary embolism were reported as occurring at stool. The physician must provide adequate laxatives to prevent this situation.

As discussed elsewhere, the acute reaction of pulmonary embolism has been thought to be due in part to spasm of the artery. This led to the use of antispasmatic substances including papaverine, aminophylline, atropine and opium derivatives, separately or in combination, as for example, spasmalgin®. Favorable results have been reported following their use, but large, well controlled series are not available. Nevertheless, it seems advisable to use these or other relaxing agents to allay the extreme tension so commonly present in these patients.

Twenty years ago serious attempts were made to develop operative procedures for removal of pulmonary emboli, but they were very hazardous and with the advent of anticoagulant therapy have been abandoned. The use of oxygen appears to be indicated in the presence of dyspnea or cyanosis to lessen the burden of the functioning area for gas exchange and hence the strain of the heart. In recent years efforts have been made to reduce the incidence of thromboembolism prior to its original development. The most popular methods have included the following:

1. Early ambulation within twenty-four hours of the operation or delivery. This is still a matter of controversy, but we incline toward the evidence in favor of it. Active proponents of this method have pointed out that merely sitting in a chair as commonly practiced is not ambulation, and indeed greater stasis may occur with the legs in dependency for long periods than if they are actively moved in bed or by walking. In ill patients we prefer the oscillating bed.

2. Deep breathing many times a day which should be immediately discontinued on any sign of thrombosis or embolism.

3. In the treatment of phlebitis we elevate the foot of the bed to speed up the flow of blood through the venous channels of the legs and pelvis, and to prevent stasis. For many years it has been our practice to do this as a prophylactic measure following surgery whenever the surgeon feels the operative procedure justifies it, and also provided the arterial supply to the limbs is adequate.[15] None of these surgical patients has had a postoperative pulmonary embolus. Torpin[12] has reported similar results, namely, no pulmonary emboli in 1,500 gynecologic operations using this simple prophylactic measure. The use of an oscillating bed in chronically ill or postoperative patients has much to recommend it. The repeated filling and emptying of the veins should aid in prevention of phlebitis.

4. Elastic stockings in the prevention of pulmonary embolism. Wilkins and Stanton[16] have recently reported that in a series of over 5,000 routine hospital admissions pulmonary emboli were significantly reduced by the continuous wearing of light elastic hose.

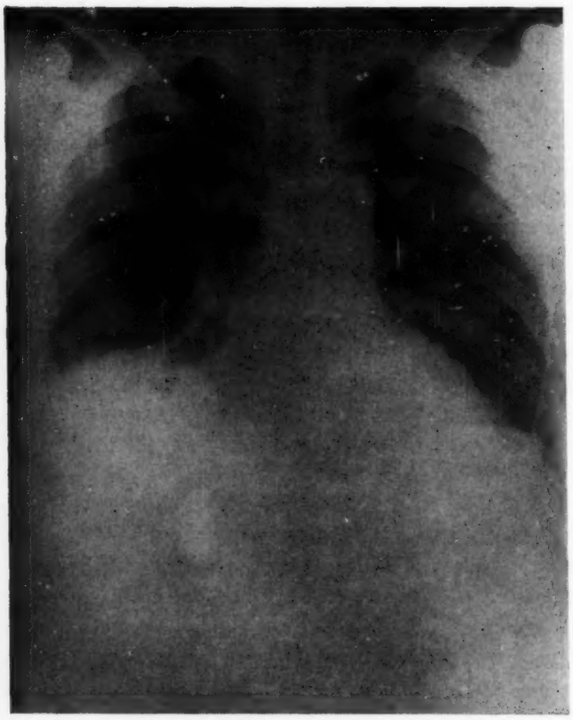


FIG. 1. Pulmonary embolus and atelectasis in a patient with postoperative phlebitis.

5. Prophylactic ligation of the veins postoperatively. This is much less popular than it was ten years ago. Some workers have even reported more emboli after ligation than in a control series. We do not favor this procedure.

6. Anticoagulant therapy on a purely prophylactic basis. We would not recommend this in all patients or after all operations, but certain situations probably justify its use according to a large series reported by Barker, Brambel and others, and our own experience. These include: (1) major pelvic surgery, (2) herniorrhaphy, (3) major abdominal surgery, especially in the presence of a history of former thromboembolic conditions, (4) surgery on older persons, (5) surgery in the presence of auricular fibrillation, coronary artery disease, or passive congestion, (6) the presence of marked varicose veins or venous insufficiency, (7) after delivery when the mother has had previous thrombophlebitis or pulmonary embolism, and (8) in all patients who have previously demonstrated a tendency toward thrombosis or embolism.

LONG-TERM ANTICOAGULANT THERAPY

The treatment of pulmonary embolism should not be limited to a consideration of the acute episode first encountered. This may be a single experience secondary to thrombosis which becomes organized and causes no further trouble, and treatment of three to four weeks' duration may be sufficient. On the other hand, the patient may continue to have repeated emboli from chronic recurrent thrombophlebitis or auricular fibrillation. We have patients who have had as many as twenty emboli from such sources. It then becomes necessary to employ long-term anticoagulant therapy. The results of such therapy are encouraging and have been reported by us.[13,14] Our experience has greatly increased since then. We have maintained more than 100 patients for from one to eight years on anticoagulants.

The following case reports present examples of interest:

CASE I. M. A. A. (N. Y. H. No. 625–583), a fifty year old man, had cholecystectomy performed because of cholelithiasis. On the eighth postoperative day he experienced sharp right chest pain and raised blood-streaked sputum. Deep phlebitis was found in his right calf. Anticoagulants were administered promptly, the leg was wrapped in warm, moist packs and elevated. An uneventful recovery ensued.

Figure 1 is a portable chest film. The right diaphragm is elevated and tented. A pleural effusion is present. There is partial atelectasis of the right lower lobe.

CASE II. Miss J. R. (N. Y. H. No. 530–436), a fifty-seven year old social service administrator, was first seen in November, 1948. During the previous ten years she had had eight illnesses that had been characterized as "lobar pneumonia." On admission she complained of cough, chest pain and blood-tinged sputum. Examination showed typical signs of mitral stenosis. X-ray of the chest showed a shadow in the right lower lung field. Dicumarol therapy was instituted and maintained for two months. The patient lapsed from treatment. She again had a sudden episode of chest pain, cough and blood-streaked sputum. (Fig. 2.)

CASE III. Mrs. E. H. (N. Y. H. No. 570–625), a fifty-one year old woman, was first seen in June, 1950, with a pulmonary infarct. At age fourteen she had had rheumatic fever and at eighteen she had been told she had a heart murmur. Until her present illness she had been entirely well. She was a business executive and led a busy professional and social life. The day

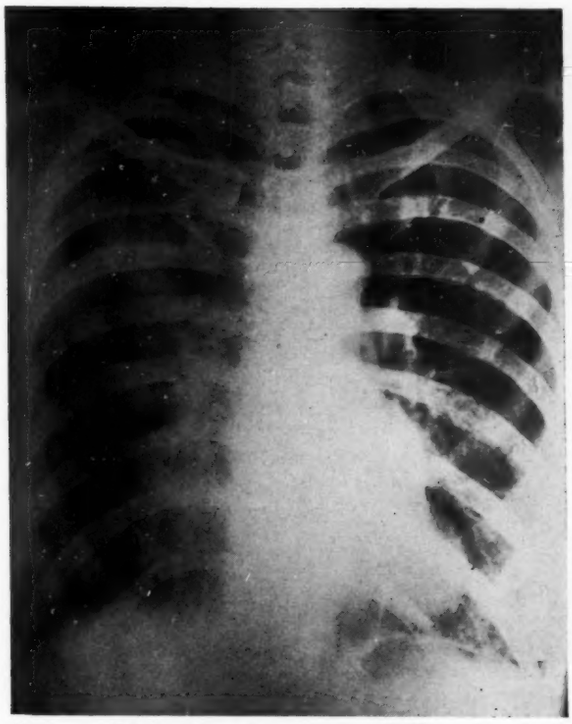


FIG. 2. Multiple pulmonary emboli and mitral stenosis.

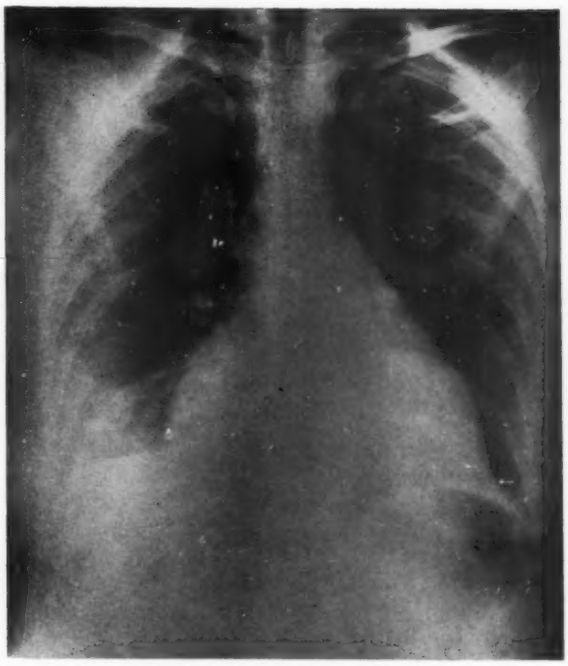


FIG. 3. Pulmonary embolus, mitral stenosis.

prior to admission she had developed severe pain in the right lower chest radiating to the back. Examination disclosed signs of mitral stenosis and aortic stenosis. There was a regular sinus rhythm interspersed with many premature contractions. The chest showed dullness and diminished breath sounds at the right base. X-ray of the chest is shown in Figure 3. This film shows generalized cardiac enlargement, bilateral pleural effusion and an increased density in the posterior basal segment of the right lower lobe which is consistent with the clinical findings of pulmonary infarction.

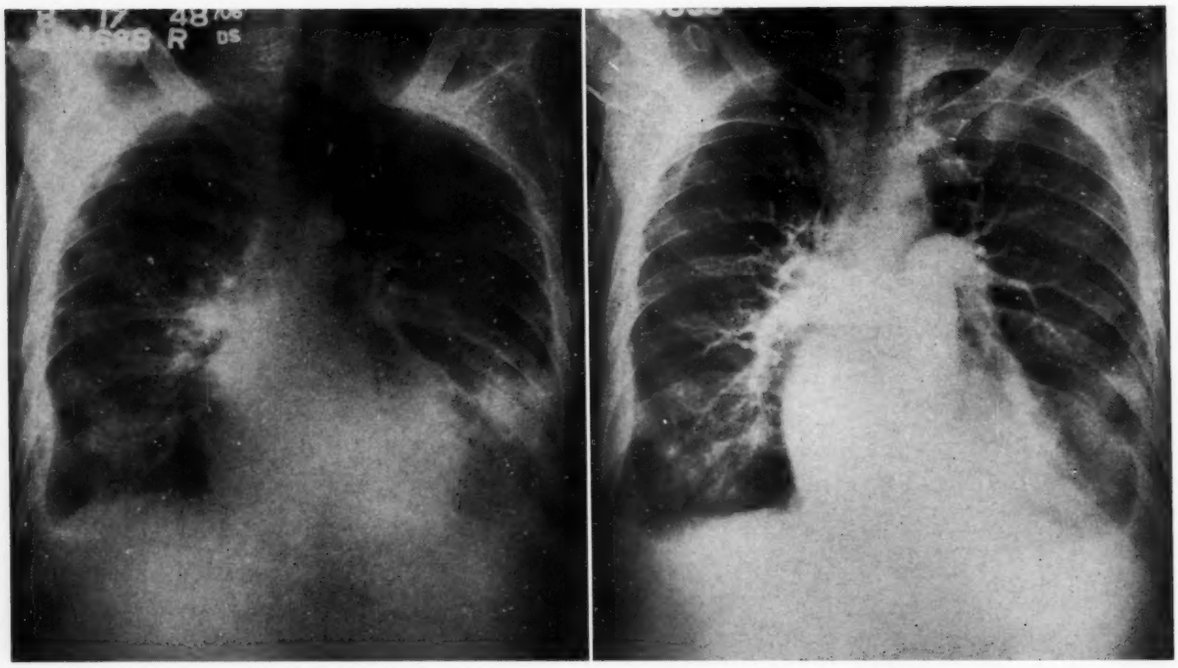
The patient ran a febrile course for a week. She was given anticoagulants starting with heparin and shifting to dicumarol. She was digitalized, and discharged on her fourteenth hospital day. Dicumarol was discontinued.

Two weeks later a second pulmonary infarct occurred. She was readmitted to the hospital and again given dicumarol. The decision was made to keep her on anticoagulants permanently. There have not been any further emboli during a period of four years. She reports every fourteen days for prothrombin determinations. She has had several minor hemorrhagic manifestations, large bruises after slight trauma, but no major ones. Her prothrombin time varies from twenty to a high of forty seconds. Her average daily dose is 75 mg. of dicumarol. While under treatment she has made two business trips to California, one to Paris and Rome and many shorter trips. Her blood tests have been arranged at various points with physicians interested and trained in this work. Her course was complicated two years ago by the development of hyperthyroidism. This was successfully controlled with radioactive iodine.

This patient represented a case of rheumatic heart disease, not fibrillating, who delivered emboli from the right auricle to the lungs.

CASE IV. L. S. (N. Y. H. No. 494–688), a forty-four year old white man, had had three previous episodes of myocardial infarction. He had also had multiple pulmonary infarctions. The x-ray of the chest (Fig. 4A) shows increased vascular markings in both lung fields and a bilateral pleural effusion. The angiocardiogram (Fig. 4B), made four and a half seconds after the intravenous injection of the contrast medium, shows the pulmonary conus, pulmonary artery and its major branches well outlined. The branches to the right lower lobe are irregular and cut off and indicate the point of lodgement of a pulmonary embolus.

The patient died several months later. Autopsy showed a large embolus in the right lower pulmonary artery branch, multiple old pulmonary infarcts and multiple myocardial infarction.



4A 4B

FIG. 4. A, pulmonary infarction secondary to myocardial infarction. B, angiocardiogram showing embolus blocking right lower pulmonary artery.

SUMMARY

The importance and frequency of pulmonary embolism has been discussed. The diagnosis and treatment have been presented. Important strides have been made in the treatment of this condition during the past twelve years since the advent of anticoagulant drugs. While death and recurrent emboli may occur during such therapy, the incidence of both has been strikingly reduced by the use of heparin and coumarin derivatives.

Greater awareness of the possibility of thrombosis and pulmonary embolism will result in earlier diagnosis and treatment and further reduction in mortality from these conditions.

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Thrombosis of Pulmonary Arteries

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MASSIVE organized thrombosis of the main pulmonary artery and its branches has for years been a recognized pathologic entity,[1,2] but it is only recently that a clinical syndrome of the disease has been evolved, making the antemortem diagnosis of pulmonary thrombosis possible.

It is sometimes astonishing how organized thrombi can occlude large branches of the pulmonary artery for periods of weeks, months or years and yet produce surprisingly little disability in proportion to the critical function of the vessels involved. Apparently this is a result of slow progression in size of the thrombi, the occurrence of recanalization and limitation in the number of large pulmonary arterial branches involved.

The syndrome of chronic massive pulmonary artery thrombosis is not a commonly encountered one, but it occurs with sufficient frequency for it to merit consideration in the differential diagnosis of mediastinal mass lesions. We have previously reported seven cases of chronic massive thrombosis involving either the main pulmonary artery, several of its major branches or both.[4] Other series in the literature bring the total of reported cases to well over 100.

Onset of massive pulmonary artery thrombosis is most commonly in pulmonary embolism arising from systemic veins or right ventricle. Thus a history of pulmonary embolism is a leading clue to the correct clinical diagnosis of the entity. Thrombosis of pulmonary arteries occurs and has been reported as a complication of certain local pathologic processes such as carcinoma of the lung, pulmonary tuberculosis, pneumoconiosis, and congenital and acquired heart disease.[3,5-7]

Cor pulmonale may antedate massive thrombosis of the pulmonary artery, or it may result from the arterial occlusion itself. In most of our previously reported cases it has not been possible to ascribe the exact pathogenesis of the syndrome.

Symptoms of chronic massive thrombosis of pulmonary arteries may have their onset in pulmonary embolism, or they may in more subtle fashion be associated with pre-existent disease of the heart, lungs or pulmonary vessels. Most often the symptoms are those of congestive failure, typically of the right heart. Duration of symptoms may be of weeks, months or years, and thrombi may exist and propagate for corresponding lengths of time. Dyspnea was a universal symptom in our previous cases. Clubbing and cyanosis were uncommon. Polycythemia was not the rule. Pulmonary embolism is common, occurring either as the initiating factor or secondarily as the result of detachment of thrombi from the pulmonary artery branches occluded. Death was in all cases sudden and unexpected.

The clinical diagnosis of pulmonary artery thrombosis is in large measure dependent upon the radiographic examination of the chest. The diagnosis is based upon discovery of enlargement involving one or more of the vessels in the hilar regions, or enlargement of the pulmonary artery itself. Thus thrombosis may involve the main artery, its primary branches and the larger branches of the latter, singly or collectively, depending upon location and extent of thrombosis. The thrombosed artery has specific fluoroscopic and radiographic characteristics. It is possessed of little motion, producing sharp, clear images on the film. The thrombosed vessel has an elliptic configuration, tapering abruptly at the distal point of thrombotic closure. Distal to the point of thrombosis the lung segment involved exhibits reduced radiability because of diminished blood flow in the pulmonary segments so affected.[8] The mass is localized to the artery or to its branches, and it is demonstrated to be continuous with those branches.

Differential diagnosis of pulmonary artery

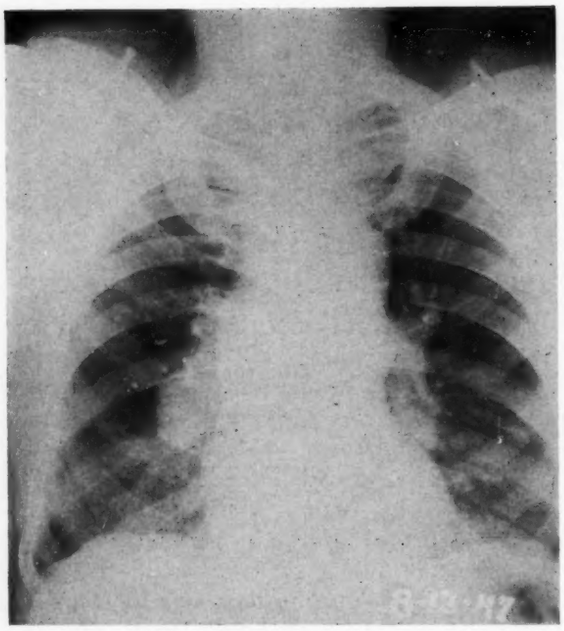

FIG. 1. Case 1. Chest roentgenogram made three weeks before death; heart not enlarged. Thrombosed right branch of pulmonary artery very sharply circumscribed, due to immobility. Similar but less obvious changes in left hilar region. Lung fields normal.

thrombosis includes the differential diagnosis of mediastinal mass lesions of variable origin, as well as other causes of pulmonary arterial dilatation. Massive thrombosis of a secondary or tertiary branch of the artery may result in a unilateral mass lesion characterized also by segmental increase in radiability of the lung, simulating check-valve obstruction of the bronchus as the result of bronchogenic carcinoma.

The electrocardiogram may be expected to reveal right ventricular hypertrophy. A change by serial records from a normal record or from left axis deviation to right ventricular hypertrophy may occur, coincident with the development of cor pulmonale. (Fig. 3.)

Angiocardiograms are of aid in the diagnosis, although as in the case of mediastinal neoplasms opacification of the mass may fail to occur. This will not necessarily be the case, especially if recanalization of the thrombus has occurred. Normal opacification of the peripheral pulmonary arteries is not to be expected.

Since our previous report of seven cases with autopsy verification[4] we have observed one additional case confirmed at autopsy. One other case in a living patient, presented herein, is not confirmed anatomically.

CASE REPORTS

CASE I. A seventy-three year old man had paroxysms of nocturnal dyspnea for two months prior to hospital admission. On physical examination there was edema of the legs, enlargement of the heart without murmurs, enlargement of the liver and shortness of breath. The blood pressure was 172/105. The electrocardiogram showed right ventricular hypertrophy. Chest roentgenograms revealed findings typical of massive thrombosis of pulmonary arteries bilaterally. (Fig. 1.) The patient improved following treatment of cardiac failure. However, relapse occurred and a few days prior to death there was clinical and radiographic evidence of pulmonary embolism. Death was sudden and unexpected. Autopsy revealed right ventricular hypertrophy. There were large thrombi firmly attached to the walls of each main pulmonary artery. The main pulmonary artery, its right and left branches, and branches to the left lower lobe, right middle lobe and right lower lobe were all occluded. Microscopically there was pulmonary arteriosclerosis and focal pulmonary arteritis. Serial chest roentgenograms indicated that thrombi had existed for at least five weeks before death occurred.

CASE II. A sixty-six year old man had a transurethral resection of the prostate gland. A preoperative chest film (Fig. 2) revealed no abnormality. During the postoperative period there was clinical and radiographic evidence of pulmonary embolism. At that time the electrocardiogram showed left axis deviation. (Fig. 3A.) Eight months later the patient returned with dyspnea and ankle edema. Clubbing of the fingers had occurred. He responded to therapy for cardiac failure. A chest film (Fig. 4) made a few days before death, eight months after the initiating episode of pulmonary embolism, revealed changes indicative of pulmonary thrombosis, and at this time the electrocardiogram showed right axis deviation. (Fig. 3B.) Death occurred suddenly about eight months after pulmonary embolism. Autopsy revealed cardiac hypertrophy involving the right ventricle especially. The right pulmonary artery was completely occluded by a laminated and firmly adherent thrombus which distended the artery to a diameter of 5 cm. and extended from the bifurcation of the artery to the middle portion of the right lower lobe. Branches of the left

FIG. 2. Case II. Control roentgenogram of chest made before operation; normal chest.

pulmonary artery to the left lower lobe were occluded at their origin. Microscopically there was slight arteriosclerosis of pulmonary arteries.

CASE III. A sixty year old woman had been dyspneic with obesity but was otherwise well until six years prior to admission, when she had her first attack of paroxysmal auricular tachycardia. Since then there had been progressive exertional dyspnea and orthopnea, and she had had mild ankle edema for several years, failure being controlled with digitalis and diuretics. On physical examination the pulse was 98, the blood pressure 130/85. She was chronically ill, with cyanosis of nailbeds and lips, and with clubbing of fingers and toes. There were palpable nodules in both lobes of the thyroid. The heart was enlarged, with a soft diastolic murmur and a systolic murmur at the apex. Venous pressure was 130 mm. of saline. There was mild pretibial and ankle edema. Circulation time, arm to tongue, was 12 seconds with ether and magnesium sulfate. The electrocardiogram revealed broad-notched mitral P waves, right ventricular hypertrophy and right bundle branch block. The hemoglobin was 18.7 gm., the red cell count 7.25 million.

Despite previous treatment for toxic nodular goiter with radioactive iodine, she continued to have attacks of tachycardia, and eight days before death she experienced onset of severe left chest pain with cough and hemoptysis. She responded poorly to supportive treatment

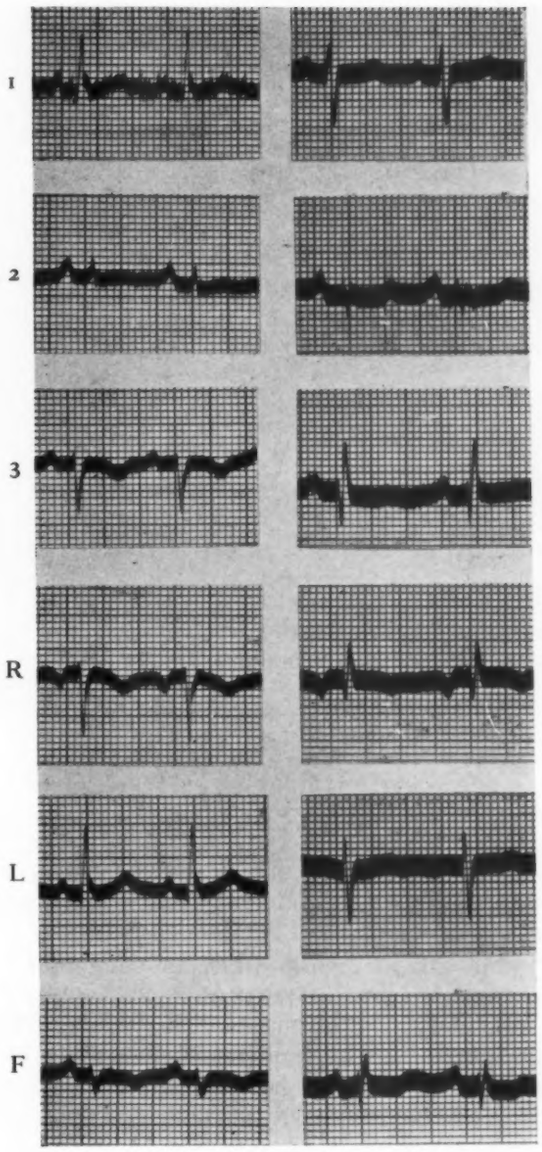

FIG. 3. Case II. A, electrocardiogram reveals left axis deviation and pulmonale P waves. B, electrocardiogram now shows right axis deviation and inverted T waves in 1, 2, 3 and V_1 to V_6. P waves are tall and peaked.

and died unexpectedly in her sleep. Autopsy revealed mitral stenosis, an atrial septal defect, and bilateral massive thrombi involving primary and secondary branches of the pulmonary artery. There was evidence of pulmonary arteriosclerosis with hemorrhage into plaques, but there was not much evidence of organization. According to morphologic estimates the thrombi had not been present for longer than one week. However, the diagnosis had been

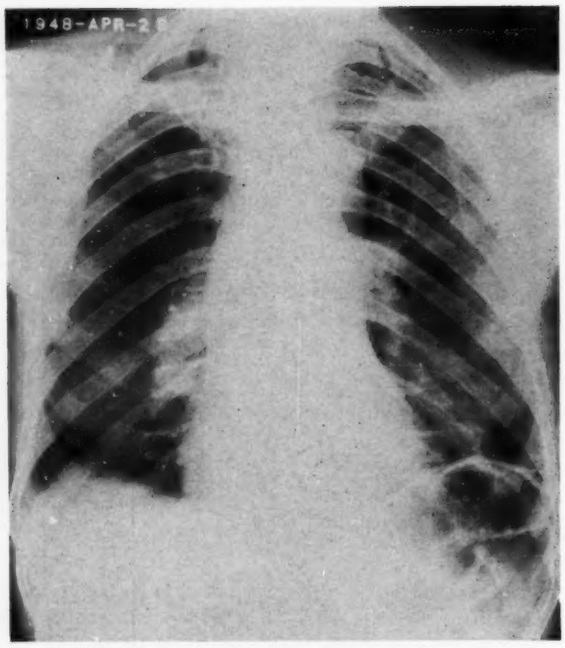

FIG. 4. Case II. Chest roentgenogram made one week before death, nine months after postoperative pulmonary embolism. Right hilus now abnormal, with mass shadow representing thrombus-filled right branch of pulmonary artery. Lung fields abnormally clear, especially the right. Minor residua of old pulmonary embolism at left lung base.

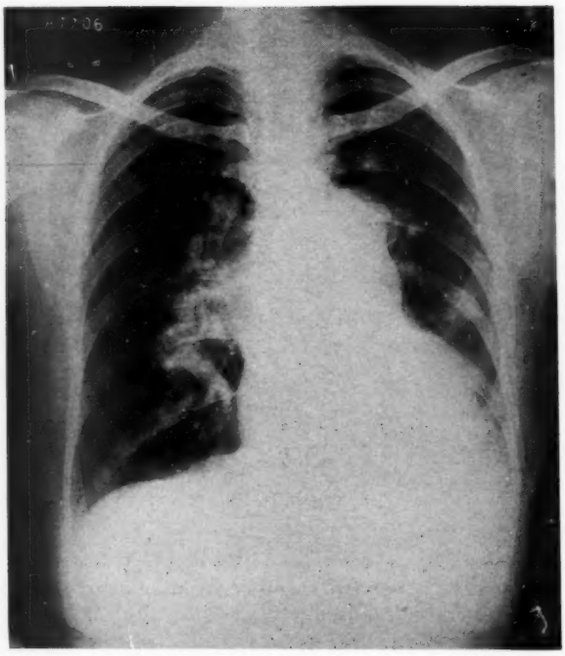

FIG. 5. Case III. Chest roentgenogram made a few days before death; cardiac enlargement. Hilar vessels huge and sharply circumscribed. Lung fields abnormally clear. Left mid- and lower lung shadows are the result of pulmonary embolism.

suspected radiographically before death, and the appearance of pulmonary artery shadows in the hilar regions had not changed much in a period of one year. (Fig. 5.)

CASE IV. This thirty-five year old man was an asthmatic, with onset early in life. Six years previously he had his first episode of decompensation, with edema of legs, thighs and trunk. He recovered but a similar episode recurred three years prior to admission. Since then, despite digitoxin and other supportive measures, there had been chronic pedal edema. Three years prior to admission there was an attack of semiconsciousness and aphasia, with right heimparesis which persisted. Diagnosis at that time was cerebral thrombosis.

On physical examination he was not acutely or chronically ill in appearance, but there was moderate respiratory distress. The blood pressure was 100/80. There was increased anteroposterior diameter of the chest, with reduced excursions and diminished breath sounds bilaterally. The heart was not enlarged and there were no murmurs. The liver was palpable 3 fingerbreadths below the costal margin, the nailbeds were dusky and there was pretibial and ankle edema. The electrocardiogram revealed right ventricular hypertrophy.

Cardiac catheterization revealed an enlarged right atrium. There was no evidence of an intracardiac shunt. Pressure in the pulmonary "capillary bed" was 18 mm. Hg., in the right branch of the pulmonary artery 96/47, in the main pulmonary artery the same, and in the right ventricle 105/5.

Hemoglobin was 16 gm., the red blood cell count 5.1 million and the hematocrit 52. The Wassermann test was negative and the venous pressure 85 mm. of saline; the circulation time arm-to-tongue was 28 seconds, arm-to-lung 9 seconds.

A chest film revealed a mass in the right hilar region, possessing all of the characteristics of massive thrombosis of the right branch of the pulmonary artery. (Figs. 6 and 7.) Comparison with a chest film made two years before had shown a normal-appearing right pulmonary artery at that time. Fluoroscopically the mass moved only slightly. The heart was enlarged a little and the lung fields were abnormally clear, in part as the result of pulmonary emphysema, but probably also in part because of reduced blood flow.

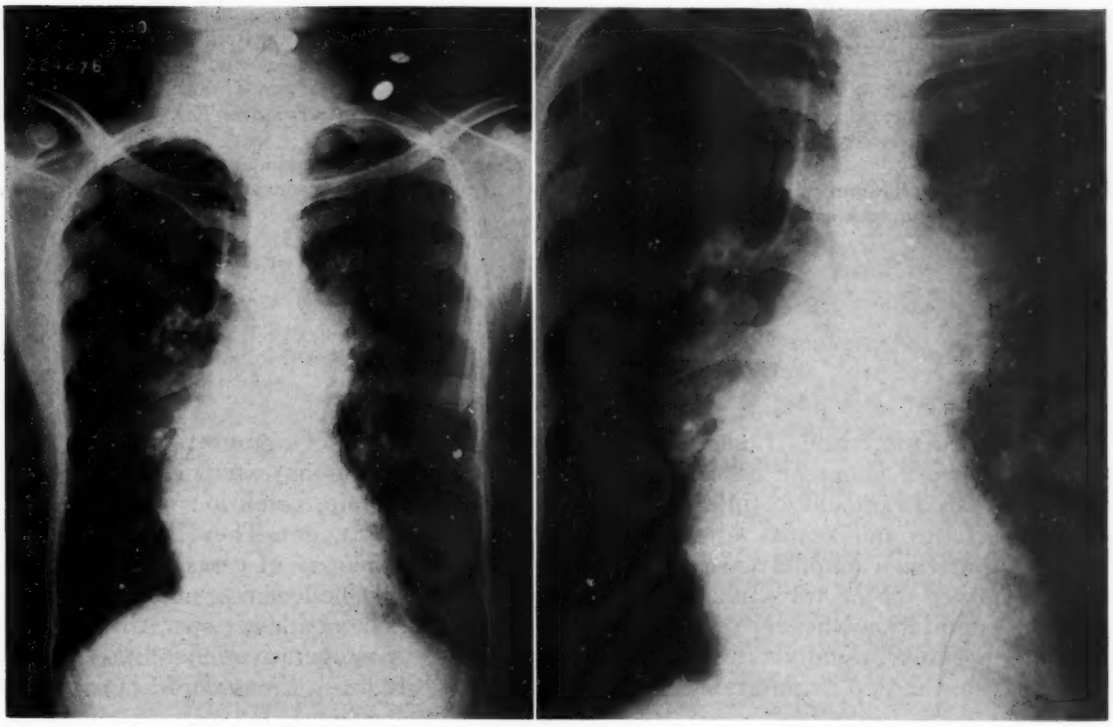

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FIG. 6. Case IV. Routine chest roentgenogram; advanced pulmonary emphysema. Pulmonary artery and its branches enlarged.

FIG. 7. Same case. Magnified view of chest roentgenogram especially to show characteristics of thrombosed artery in right hilar region. Note sharp, smooth elliptic lateral border of mass and its abruptly tapered lower extremity. Left hilar mass composed of main pulmonary artery and its left branch. Abnormally clear lung fields as the combined result of pulmonary emphysema and reduced blood flow.

Angiocardiograms did not show appreciable opacification of the arterial circulation of either lung. The main pulmonary artery and its primary branches opacified faintly, and there was a suggestion of canalization of the right branch. Previous passage of a cardiac catheter into the right branch of the pulmonary artery seemed to support the impression of recanalization. The patient was discharged unimproved.

SUMMARY AND CONCLUSIONS

Large thrombi may exist in the pulmonary artery, its branches or in both for long periods of time. Often they become organized and recanalized. They arise either locally or are the result of emboli which originate in systemic veins. Symptoms are usually those of congestive heart failure, typically of the right heart. Cyanosis, clubbing and polycythemia may occur but they are not the rule. Dyspnea is a frequent symptom. Electrocardiograms uniformly reveal right ventricular hypertrophy. Pulmonary embolism may initiate the syndrome, and as an episode in the course of the disease it is a common occurrence. There are characteristic roentgenographic findings which are to be differentiated especially from mediastinal mass lesions of variable etiology, notably carcinoma with check-valve obstruction of the bronchus. Death is usually sudden and unexpected.

Acknowledgment: Histories and illustrations of Cases I and II are reprinted from the *American Journal of Roentgenology, Radium Therapy and Nuclear Physics*, Volume 69, February, 1953,[4] with permission of the Publisher and the Editor.

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Silbert and Haimorici recommend that the site of amputation for ischemic gangrene be as low as possible, depending upon extent of this process, amount of infection present, severity of pain, condition of the arteries and tissues adjacent to the gangrenous area, etc. They believe that thigh amputations are indicated only in a minority of cases. They prefer toe or transmetatarsal amputation, or supramalleolar or mid-leg amputation whenever possible, as these procedures have a lower operative mortality, usually a less painful stump and better postoperative rehabilitation than do amputations through the thigh. (*Richard A. Leonardo, M.D.*)

Foreign Bodies in the Bronchopulmonary Tract

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THE basic understanding upon which modern diagnosis and treatment of pulmonary disease is founded had its origin in the experience gained in the solution of foreign body problems. From these observations came the explanation of the mechanical features which produce the wheeze, obstructive emphysema and atelectasis. The relative incidence of foreign body problems becomes smaller and smaller as this field of medicine progresses. This is due in part to prophylaxis but more so to the wider application of bronchoscopy in the diagnosis and treatment of all pulmonary diseases. Today foreign body work is a small part of the bronchologist's practice. However, the principles involved in these problems are basically important and can be applied to all phases of pulmonary disease.

This subject cannot be covered without first acknowledging the main source of knowledge and authority. From the pioneer work of Chevalier Jackson, carried on by his associates and students, has come a vast storehouse of information.[1–6,8] Any treatise must of necessity refer to these works and merely add the author's own interpretation and emphasis to the various aspects of the subject of foreign bodies in the bronchopulmonary tract.

Etiology. Age appears to be the most significant factor in the etiology of foreign bodies.[1,4] By far the greatest number of cases occur in children.

Size and shape of the foreign body help determine its location and symptomatology. Those too large or irregular to pass through the larynx will either lodge in the hypopharynx or enter the esophagus. Occasionally they will lodge in the larynx, usually precipitating an acute respiratory emergency because of resulting laryngeal obstruction. As the foreign bodies become smaller, they will lodge further down in the tract. Size and shape determine whether they will pass through the larynx, since the glottic chink has its greatest measurement in the anteroposterior diameter with a relatively small width, greatest in the posterior aspect.

Carelessness on the part of adults contributes to the problem not only for themselves but also for the children under their care. This is particularly true of foreign bodies held in the mouth, a practice which is quickly mimicked by children. In the older age groups physical disability and senility contribute to the problem.

To list all of the various types of foreign bodies is virtually impossible. In the many detailed analyses published[1,2,4–6,8] it is apparent that vegetal foreign bodies are the most numerous and occur principally in children. While bones usually lodge in the esophagus, those that do enter the respiratory tract are commonly overlooked. Grasses are rare but their ratchet-like action which drives them deeper and deeper into the bronchi makes them a difficult problem for removal. Metallic foreign bodies such as screws, pins and nails are common. Dental plates are uncommon but teeth are frequently seen. Coins are rare since it is easier for them to pass into the esophagus than into the trachea because of the necessity of turning from the coronal plane to the sagittal plane to get through the glottic chink. Endogenous foreign bodies, such as broncholiths, are rare. Of those foreign bodies which do enter the bronchopulmonary tract, the highest number lodge in the bronchus, next in the trachea and the smallest number remain in the larynx.

Pathologic Physiology. The mechanism of partial and total obstruction of the trachea or bronchi is basic to the understanding of the action of foreign bodies in the bronchopulmonary tract.

Chevalier Jackson has best described it as follows:[4–6] *Bypass-valve* action occurs when the foreign body is of a size and shape or lodged at a point where air can pass by it on both inspiration and expiration. Since there is no obstruction to the flow of air, there will be no change in the x-ray picture and very little will be found on physical examination other than possibly a wheeze.

As the air space around the foreign body decreases due to edema of the mucosa or to the further progress of the foreign body into the smaller bronchi, the *check-valve* phenomenon occurs. This is based on the change in the lumen of the bronchi with enlargement during inspiration and decrease during expiration. As a result, when the air space reaches a point where air can enter on inspiration but is trapped on expiration, the picture of obstructive emphysema occurs. When the foreign body lodges at a point where air can leave but not enter, a *ball-valve* action results in atelectasis. This phenomenon is occasionally intermittent, which complicates diagnosis.

The final stage is the *stop-valve* phenomenon where neither inspiration nor expiration results in air exchange and the portion of lung involved becomes completely atelectatic.

There are two other basic mechanisms which should be described. Holinger explained them clearly in his paper on bronchial obstruction.[3] The first is the mechanism by which subcutaneous emphysema is produced. Partial obstruction of a bronchus which produces obstructive emphysema causes an increase in the alveolar pressure. This may be enough to form bullae beneath the visceral pleura. Air from rupture of these bullae will travel along the subpleural space to the hilus of the lung, then up the mediastinum to the neck and out under the skin. Rupture of the bullae into the pleural cavity results in a sudden pneumothorax. This usually precipitates an acute emergency calling for needle aspiration and a closed pneumothorax drainage system.

The second pathophysiologic change has to do with the effect of bronchial obstruction on the cardiovascular system. There is increased pulmonary capillary blood pressure resulting in transudation of serum into the alveolar spaces. An increase in intrabronchial and intra-alveolar negative pressure causes exudation into the alveoli. The combination of the increased negative pressure and the increased pulmonary capillary blood pressure results in pulmonary edema and circulatory failure.

DIAGNOSIS

The first step toward accurate diagnosis is a high index of suspicion coupled with a careful history. In many cases a positive history of foreign body can be elicited, but there are enough cases in which the incident occurred where the patient was unaware of it due to sleep, coma, anesthesia, etc., or the patient was too young to give a history and the incident occurred out of sight or sound of the responsible party. Thus there is need for a high index of suspicion on the part of the physician when confronted with a complaint or physical finding in the bronchopulmonary system which appears to have an "obscure etiology."

A review of actual case histories shows that many physicians refuse to accept the patient's story of aspiration of a foreign body.[9] The patient's plea for x-ray examination is disregarded and this, coupled with a superficial physical examination, results in the patient's being sent back home to await the inevitable sequelae which follow the long lodgment of a foreign body in the bronchopulmonary tract, i.e. sepsis, suppurative lung disease, decreased pulmonary function, disability and even death.

The first physician seen is responsible for the early diagnosis or delay, depending upon his awareness of the need for complete study including x-ray and diagnostic bronchoscopy.

Usually there will be an episode of coughing and choking associated with varying degrees of respiratory distress. This is often followed by a symptomless interval in which the cough becomes negligible or disappears. During this interval there is little or no respiratory distress, and it is not until edema, infection and suppuration appear that symptoms of cough, wheeze and dyspnea recur. The patient then presents his problem to the physician for solution. The original episode may have been completely forgotten. It is this symptomless interval which gives a false sense of security to the patient and doctor alike, and delays diagnosis and treatment.

Keeping in mind the basic pathologic physiology and anatomy, it is not difficult to bring to mind the symptomatology and physical signs found in these problems. They vary with the location, size, shape and length of lodgment of the foreign body.

The foreign body in the larynx produces hoarseness, pain, dysphagia, cough and often stridor.

When the foreign body is in the trachea and freely movable, the patient will describe a slapping on inspiration and expiration. The examiner will hear at the open mouth what Jackson has called the "audible slap" and feel the "palpable thud" as the foreign body is trapped at the larynx by the vocal cords during cough or forced expiration. A wheeze, when present, is best heard at the open mouth. It is usually heard when the foreign body lodges somewhere in the trachea and becomes stationary. The cough is often "brassy" in character. Actual stridor may be present. The larger the tracheal foreign body and airway obstruction, the greater will be the inspiratory effort with associated retraction of the suprasternal areas and intercostal spaces. C. L. Jackson has stressed the importance of bilateral emphysema as a sign in tracheal foreign bodies and one which is frequently overlooked. It is often confused with bilateral bronchial obstruction due to multiple foreign bodies.

Foreign bodies in the bronchi result in cough, wheeze, dyspnea and the wide variety of signs and symptoms previously discussed. They are those seen in the various stages of bronchial obstructions and resulting distal pulmonary disease.[7] X-ray studies, including fluoroscopy, inspiration and expiration films, are valuable.

Partial obstruction resulting in obstructive emphysema causes limited chest expansion, hyper-resonance and decreased or absent breath sounds on the involved side. A wheeze is usually heard over the involved bronchus but may be transmitted over all of the chest. Fluoroscopy will show the mediastinal swing to the uninvolved side on expiration plus the flattened diaphragm on the involved side.

With complete atelectasis there will be decreased to absent breath sounds, dullness to percussion and decreased expansion on the involved side. No wheeze is heard. Fluoroscopy will not show a mediastinal swing. Films will show shift of mediastinum to the atelectatic side which is constant on inspiration and expiration. The diaphragm will be elevated on the involved side. There may be compensatory emphysema of the uninvolved side which may confuse both the radiologist and bronchoscopist as to the true location of the foreign body.

Appropriate film studies will also disclose other mediastinal abnormalities, esophageal pathologic conditions and paravertebral disease. Additional diagnostic x-ray aids include planigraphy and bronchography. As is apparent from our previous discussion, the radiologist's report may be completely negative, and diagnosis and treatment must depend entirely on history and physical examination.

The differential diagnoses must include all of the diseases of the bronchopulmonary system. This symposium is dedicated to this problem. As Jackson has said, "A wheeze may be anything from peanuts to cancer." In addition to the primary diseases of the bronchopulmonary tract, obstructive phenomena in the esophagus, whether by foreign body or other disease such as diverticulum or tumor, may compress the trachea and simulate a tracheal foreign body. Cardiovascular anomalies are of growing importance today and are increasing as a differential diagnostic factor in bronchopulmonary disease. Compression of the trachea or bronchi by an anomalous vascular structure will produce any of the basic obstructive features already described.

TREATMENT

The treatment of foreign bodies of the bronchopulmonary tract is, with very few exceptions, removal via the natural pathway by peroral bronchoscopy. Jackson reports that only 2 to 4 per cent will be coughed up spontaneously.

The instrumentarium is essentially that needed for diagnostic and therapeutic bronchoscopy in general. All that should be added are the few basic foreign body forceps peculiar to the individual problem at hand. A manikin board is necessary for practice. Magnets should be added for ferrous foreign bodies. However, it is not within the scope of this paper to go into the detailed problem of foreign body extraction. For this information the reader is referred to the basic textbooks on peroral endoscopy which thoroughly and adequately cover this subject.[4–6] To repeat this information here would be at best inadequate and superfluous.

There are certain aspects of treatment which do logically fall into the scope of this paper and which bear repetition and re-emphasis. Foreign body extraction should not be attempted with inadequate instrumentarium unless an impending fatality necessitates heroic action. Any-

thing else must be considered "meddlesome" and invariably complicates the final solution.

Where the location of the foreign body is in the distal segmental bronchi beyond endoscopic vision, biplane fluoroscopic guidance is necessary.[5,6] If this is not available, the patient should be referred to an institution with such facilities. With the use of this special x-ray equipment the percentage of foreign body removal rises to 99 per cent. There is certainly no justification for thoracotomy and possible lobectomy for removal of such foreign bodies because this equipment is not available in one's own institution.

The problem of treatment or management varies with the location and characteristic of the foreign body. Laryngeal foreign bodies demand immediate attention. Care must be taken to avoid dislodging it into the trachea or doing irreparable damage to the larynx. Although the great majority of foreign body extraction is best handled under local anesthesia, general anesthesia may be preferred in problems where absolute immobility of the patient at the time of application of the forceps and extraction is necessary. Tracheal foreign bodies, too, demand immediate attention. At any moment complete obstruction of the airway may occur with asphyxiation of the patient.

Occasionally edema of the larynx or an irregular shape to the foreign body will not allow its return via the larynx. This will require tracheotomy for adequate room to remove it.

It is generally true that the sooner the foreign body is removed, the easier the solution and the quicker the recovery. However, in problems where impending asphyxia or other complications are not of major concern, it is better to complete a thorough and adequate study, assemble the proper instruments, stabilize the patient's physical status at the highest possible point and then proceed with bronchoscopy. A duplicate foreign body should be obtained and removal practiced on the manikin board before it is attempted on the patient. This is much better than rushing into such a problem with inadequate study and preparation, improper instruments and, above all, assistants who are not acquainted with the problems involved. Do not allow yourself to be forced into a situation where your better judgment tells you that it is not the proper thing to do.

While the laryngeal and tracheal foreign bodies are usually obvious on bronchoscopic examination, the bronchial foreign body may be obscured by edema, stenosis, granulation tissue or secretions. Repeat bronchoscopies are indicated in these circumstances. Often it is only after resolution of the infection and establishment of proper drainage that the foreign body becomes apparent.

On occasion a non-opaque foreign body will require thoracotomy and resection for final diagnosis and treatment, the operative diagnosis being the end result of a long-standing foreign body, i.e., lung abscess, stenosis of the bronchus or bronchiectasis. Also, the opaque foreign body beyond the reach of the costophrenic bronchoscope and biplane fluoroscopic technic will require thoracotomy for removal.

Not all foreign bodies enter via the larynx. Penetration through the chest wall may result in a problem where the location is such that it can be removed via the bronchoscope with biplane fluoroscopic assistance. More often thoracotomy is required for removal.

PROGNOSIS

In the routine uncomplicated foreign body problem, properly handled, extraction and uneventful recovery should be expected. However, a certain number of cases terminate fatally for reasons beyond the power of the physician to circumvent; for example, the tracheal foreign body which suddenly obstructs while the patient is en route to the hospital, or perforation of the trachea or bronchi with mediastinitis which does not respond to chemotherapy and surgical drainage. Fulminating tracheobronchitis and suppurative lung disease may cause extensive morbidity and mortality. Vegetal foreign bodies such as peanuts cause severe mucosal irritation and will cause obstruction due to secretion, edema and granulation tissue. However, many of the complications and sequelae are tied up to late diagnosis. These factors can and should be prevented.

All foreign body problems should be carefully followed up postoperatively. In the immediate postoperative period the problem of infection, respiratory embarrassment due to obstruction, or mediastinal complication should be recognized early. Immediate treatment is necessary to prevent unnecessary morbidity or mortality.

Postoperative x-rays are necessary in all cases whether symptoms are present or not.

Persistent physical findings and/or x-ray findings indicate the need for careful follow-up with repeat bronchoscopy and occasionally bronchograms. There may be a residual fragment of foreign body or a granuloma, stenosis or bronchiectasis which requires active treatment.

It is generally true that the lungs have unusual recuperative power. The vast majority of foreign body problems become completely asymptomatic even in the face of roentgen evidence of residual bronchiectasis. Asymptomatic bronchiectasis of foreign body etiology does not warrant resection on roentgen evidence alone. However, there are a certain number of cases with active suppurative disease requiring resection of the involved segment.

SUMMARY

1. From the pioneer work in foreign body problems by Chevalier Jackson, his contemporaries and students, has come a fundamental understanding of the pathologic physiology of bronchopulmonary disease.

2. Foreign bodies must be considered in every differential diagnosis of bronchopulmonary disease. This is particularly true in those cases with an obscure etiology or in those cases in which response to treatment is not what experience has taught you to expect.

3. The most important factor in the incidence of foreign bodies is carelessness on the part of the patient or the responsible adult. Children are the greatest offenders and should be carefully supervised in their eating habits and play. Vegetal foreign bodies are most commonly found.

4. A positive history with negative physical examination and x-rays demands diagnostic bronchoscopy. The symptomless interval is misleading.

5. Foreign body extraction should be carried out only after proper study and preparation, and with adequate instrumentarium and assistance unless asphyxia or fatality is imminent.

6. The vast majority of foreign bodies can be removed successfully via peroral bronchoscopy with complete asymptomatic recovery.

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Traumatic Injury to Lung

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A CONSIDERATION of trauma to the lung cannot be disassociated from trauma to the thoracic wall and pleura, which almost invariably accompanies it. Normal respiratory function is as dependent upon the integrity of the latter structures as upon the integrity of the lung itself. The intimate interrelationship of these three structures in carrying out the mechanical aspects of the vital function of respiration is the main characteristic which distinguishes this class of injuries from trauma elsewhere in the body.

PATHOLOGY AND PATHOLOGIC PHYSIOLOGY OF LUNG TRAUMA

The physiologic alterations in respiratory function accompanying lung trauma are referable basically to: (1) Exudative or transudative reactions involving the tracheobronchial tree, respiratory epithelium and pulmonary interstitial tissue; (2) breaks in the anatomic integrity of the lungs, pleura and chest wall and their sequelae; (3) Traumatic and hemorrhagic shock and their sequelae.

When lung tissue is injured it reacts with the same processes of hyperemia, transudation, leucotaxis and exudation, and localized or diffuse necrobiosis which characterize the inflammatory reaction in other soft tissue to a variety of traumas. The type and severity of the reaction will depend upon the character and intensity of the inciting agent. This agent may be trauma of many types. A general classification follows:

(1) Gross physical trauma: Blast, contusion, penetrating missiles, etc.; (2) radiation effect: Thermal burns, x-ray, gamma radiation, etc.; (3) chemical substances: Irritant gases, smoke, smog, etc.; and (4) bacteria and toxins.

The initiating factor of the inflammatory reaction, as elsewhere, appears to be an irritant sufficient to damage tissue and release certain tissue substances which stimulate a state of hyperemia, capillary dilatation and fluid and leukocyte transfer into the area involved. Even at an early stage in this process in the lung, a critical physiologic derangement may develop. This is because any exudative or transudative reaction involving the alveolar or interstitial pulmonary tissue immediately imposes a physical barrier interfering with the transfer of gases from the alveoli across the respiratory epithelium into the capillaries and vice versa. When this derangement is localized, the reserve of pulmonary functioning tissue elsewhere in the lung prevents critical encroachment upon respiratory function. When this process is widespread, however, even at this early stage, asphyxia will be precipitated when the respiratory reserve is not sufficient to carry the patient's metabolic needs.

Following the early stage, the progress of the inflammatory reaction will depend upon the presence of severe, prolonged or repeated traumatic insults, the presence of secondary bacterial invaders and the factors of tissue or serologic (immunologic) resistance to the bacterial invaders and toxins. The early reaction may progress to a pneumonitic picture, with the infiltration of leukocytes and other cellular elements and a deposition of fibrin.

When there is sufficient tissue damage, either primary (due to the initiating trauma) or secondary (due to an intense inflammatory or bacterial effect), lung necrosis will develop. This may liquefy, forming an abscess. The abscess may resolve or evacuate itself endobronchially, or erupt into the pleura forming an empyema. The size and location of the process, in addition to the other factors previously cited, will determine the eventual course.

In the process of healing the elaboration of fibrous tissue in injuries sufficient to produce tissue destruction is the rule. The extent and intensity of the fibroplastic process depends upon the character and duration of the inflammatory reaction. Evidence that there is regeneration of pulmonary alveoli after destruction

is inconclusive and the weight of evidence is strongly against this.[1–4]

Within the pleural potential space there is a pressure which is negative in reference to atmosphere. This is the result of the elastic recoil of the lungs pulling upon the collapsed, airtight space within the relatively unyielding bony thorax. This pressure negativity is transmitted throughout the chest and has its effect upon the intrathoracic organs. Regular, cyclic variations in the amount of negativity produced by the diaphragm and other muscles of respiration result in the inflow and outflow of pulmonary air and assist in the filling of the great veins and the heart. During normal, quiet respiration, inspiration is accomplished by contraction of intercostal and diaphragmatic muscles, raising the costal arcs and depressing the diaphragm, thereby actively increasing the intrathoracic volume. Expiration under this circumstance is passive, occurring when these muscles relax. When respiration is more active (tachypnea, hyperpnea dyspnea), or when there are exaggerated movements such as sneezing or coughing, expiration also becomes an active phase with chest, neck and abdominal muscles brought into play. Derangement of this balanced system may be caused by pneumothorax or hemothorax, conditions resulting when the pleural cavities contain air or blood, or by an instability of the chest wall.

Breaks in the anatomic integrity of the bronchi and other air-containing element of the lung with an intact visceral pleura may give rise to pulmonary interstitial emphysema (P.I.E.).[5,6] If this accumulation of air breaks through the visceral pleura, pneumothorax results. If the trachea, bronchi or primary bronchioles are involved in the injury, or if there is dissection of the air centrally along the course of the bronchi and pulmonary blood vessels, mediastinal emphysema will result. (Fig. 1.)

Mediastinal emphysema, regardless of etiology, is a grave complication for two principal reasons: first, because of the production of a mediastinal pressure which prevents adequate filling of the great veins and heart and is rapidly fatal; and second, because of the frequent accompaniment of mediastinal infection which by pressure and sepsis rapidly produces death. The physical findings accompanying the development of mediastinal emphysema are characteristic and it is important to recognize them early so that definitive measures may be taken without delay. Frequently there is a feature of the history other than trauma to the chest, such as foreign bodies in the esophagus or respiratory tree and recent

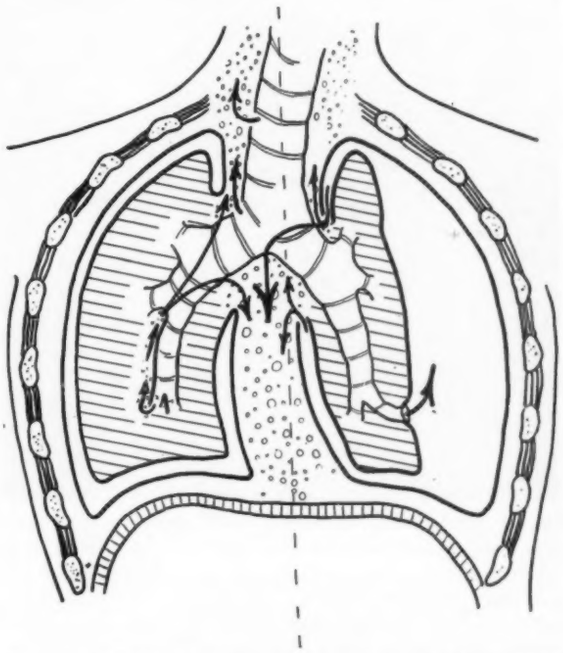


FIG. 1. Diagrammatic cross section of chest, with arrows indicating routes by which air from the respiratory tract may form mediastinal emphysema; on the left, from the trachea, bronchus and from pulmonary interstitial emphysema traveling centrally along the bronchovascular structures; on the right, from pneumothorax by leaks in the pleura in the superior and inferior hilar regions.

esophageal or tracheobronchial endoscopy, which will lead us to a high index of suspicion of this development.

Early, the patient will complain of substernal (or subxiphoid) pain which is aggravated by swallowing or breathing. At this time auscultation may reveal a precordial crunching sound synchronous with the heart beat (Hamman's sign). There may also be guarding of the movements of the neck. There is then noted crepitus on palpation of the cervical, presternal or epigastric regions, and distention of the neck veins and progressing degrees of circulatory failure as the pressure on the great veins grows, extending into frank shock, collapse and death. This progression may occur very rapidly, within four to eight hours. With it may be an accompanying dyspnea and cyanosis

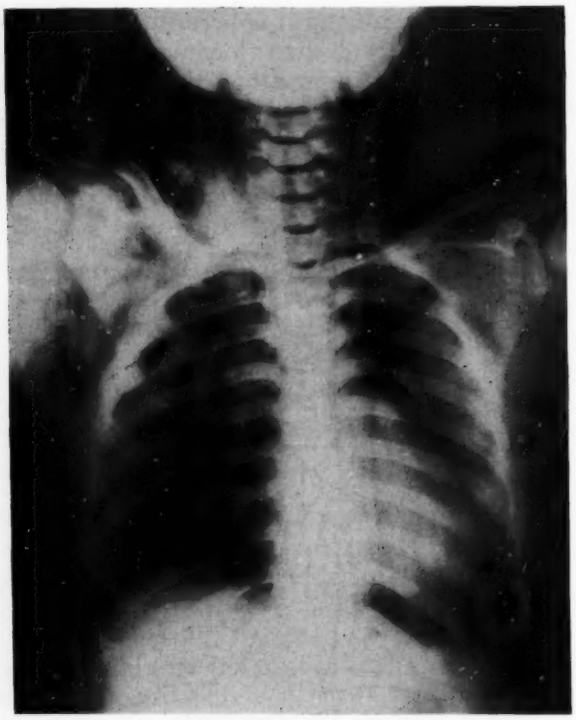


FIG. 2. X-ray film showing mediastinal emphysema which developed in a twelve year old girl within minutes following perforation of a foreign body through the left main bronchus. Air streaking and widening of the mediastinum and air dissecting up into the fascial planes of the neck are well seen.

which reflect the effect of circulatory failure, more than the pressure on the trachea and bronchi. These latter structures are semi-rigid and maintain their patency after the mediastinal pressure has obstructed the great veins.

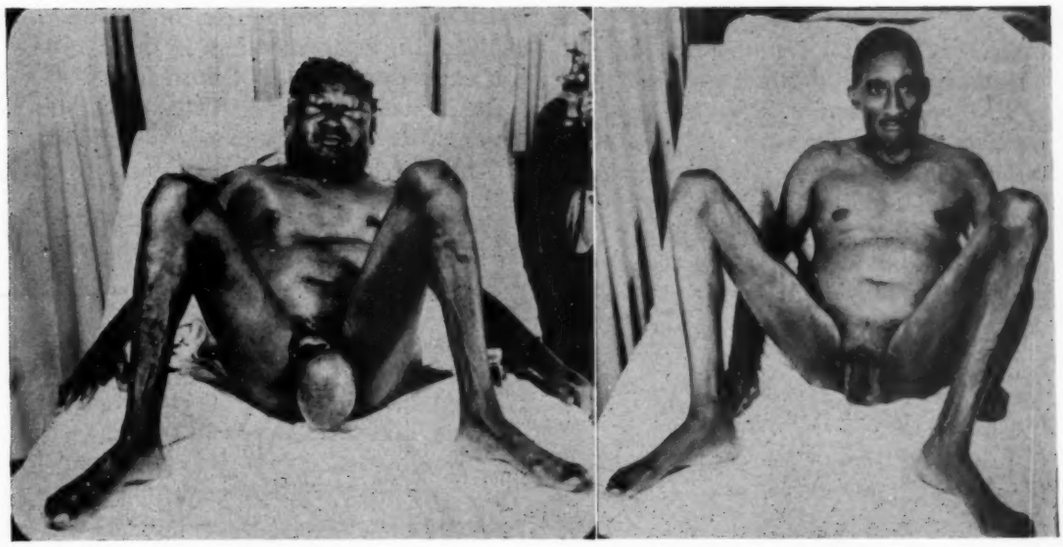
A prominent feature is the voice change which occurs due to pressure and tension on the recurrent laryngeal nerves. The sound is metallic, strained and slightly hoarse; a nasal quality is also characteristic. In some cases the terminal event may be signs of progressing sepsis. Sepsis may occur in spite of surgical decompression of the mediastinum, if the mediastinum has been heavily seeded with bacterial organisms from the site of leakage. An early diagnosis is made by x-ray which will show the streaking of air in the mediastinum and a widening of the mediastinal shadow. (Fig. 2.) A lateral film is particularly valuable since retrosternal air will be seen clearly.

Rupture of interstitial emphysema through the visceral pleura or the direct or indirect communication of a bronchus, bronchiole or alveolus with the pleural cavity, causes what is called *closed* pneumothorax. Pneumothorax may also follow rupture of mediastinal emphysema into the pleura.[7] If the penetration comes through the parietal pleura with a communication of the pleural space to the outside through the chest wall, or indirectly through the diaphragm and abdominal wall (as occasionally seen during abdominal surgery), *open* pneumothorax is said to exist.

When a defect in the parietal pleura is present with pneumothorax, air frequently escapes into the subcutaneous tissue producing subcutaneous emphysema. Alarming swelling of the subcutaneous tissues will occur if there is persistent leakage through the parietal pleural defect, or if there is communication with the bronchial passages through a zone of pleural symphysis at the place of penetration. However the swelling, which may extend from the face down to the legs along the fascial planes, is essentially innocuous, giving rise to no great danger or discomfort. (Fig. 3A and B.)

The collapse of one lung resulting from pneumothorax ordinarily can be compensated for by the ventilatory capacity of the remaining lung. However, a dangerous situation arises when there is a progressive increase of air with a build-up of tension within the pneumothorax cavity. When the visceral pleura or chest wall defect acts as a valve, permitting air to enter the pleural cavity during one phase of respiration and not permitting escape during the other, successive increments of air build up the intrapleural tension. In closed pneumothorax forced expiration against a closed or partially closed glottis, as in coughing, pushes air through the visceral defect into the pleural space under great pressure. The same events may follow rupture of an emphysematous bulla or bleb. In open pneumothorax tension will develop when the chest wall defect allows air to enter during inspiration and does not permit escape during expiration. (Fig. 4.) This repetitious build-up of pressure completely collapses the lung on the affected side, displaces the mediastinum away from the affected side, angulates and tends to collapse the great veins and seriously interferes with cardiac filling. With the mediastinal displacement the opposite hemithorax is encroached upon and the ventilatory capacity of the remaining lung is lessened. Unless relieved promptly, the condition results fatally.

In open pneumothoraces, with external openings smaller than the glottic opening and without the valve-like phenomenon of the wound, there is collapse of the lung on the affected side,



3A 3B

FIG. 3. A, photograph of patient admitted because of progressive subcutaneous emphysema. Eight years previously lobectomy had been performed for tuberculosis of the right upper lobe, followed three weeks later by apical thoracoplasty. Subsequently the patient was symptom-free and repeated tests for tuberculosis were negative. At the present admission x-rays and complete work-up revealed no evidence of the cause of this condition. B, patient after spontaneous subsidence of the condition two weeks later, with no specific therapy. There was no recurrence of this condition or tuberculosis.

but no development of tension pneumothorax. An open wound exists through which air both enters and escapes, and there is a to and fro motion of the mediastinum to some degree, but the patient may stabilize in spite of these conditions.

In open pneumothoraces with external openings larger than the glottic aperture itself, the volume exchange occurs less from the lung through the trachea and glottis with the respiratory effort, but more from the pneumothorax through the chest wall defect. This extrapulmonic exchange which is proportional to the relative size of the glottic and chest wall openings obviously cannot effect aeration of blood. Meanwhile the intrapulmonic air merely swings from one lung to the other ("Pendelluft Phenomenon"), in association with swinging or fluttering of the mediastinum back and forth. In addition to the failure of respiratory effort, the to- and fro-shift of the mediastinum exerts a great interference with the return of venous blood to the heart by angulating the venae cavae. Here again a fatal issue rapidly supervenes unless this condition is relieved.

When the normal semi-rigid structure of the thorax is destroyed by a crushing injury, respiration is reflected in flail-like, paradoxic motion of the unstable portion of the chest wall. This occurs, for example, when a number of adjacent ribs are fractured in two or more places, thereby detaching the intervening segments from the bony rigidity of the remaining thorax. These segments then passively sink in

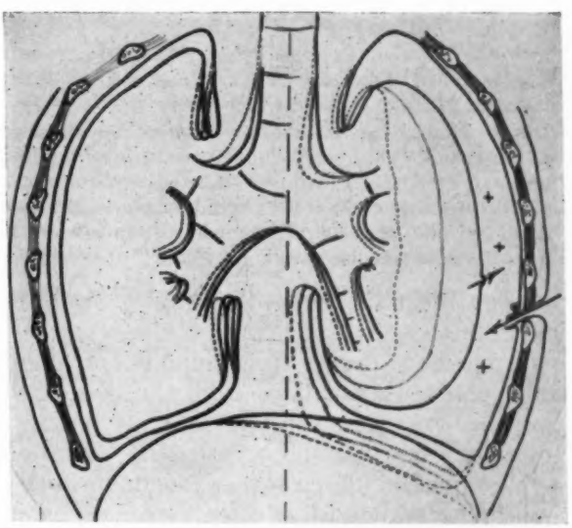
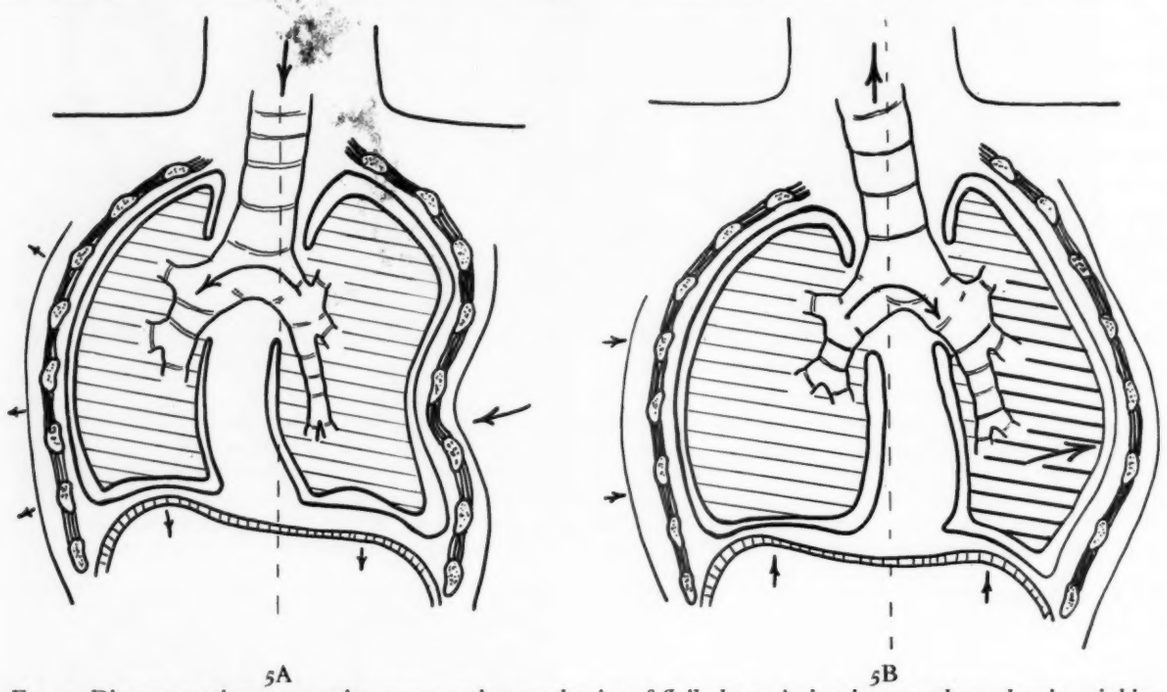


FIG. 4. Diagrammatic cross section of chest representing the stages of development of tension pneumothorax. Progressive collapse of lung, shift of the mediastinum and depression of the diaphragm on the affected side are seen. This follows the intake of air through a pulmonary or chest wall defect during one phase of respiration and failure to release this air during the other phase. Increasing increments of air pressure are added to the pleural space, as through a valve, resulting in this condition.

with inspiration and move outward with expiration. (Fig. 5.) This diminishes the efficiency of the respiratory effort and wet lung rapidly develops. When a large area of the chest wall is unstable, or where both chests and the sternum, or both, have been crushed, the paradox must be stopped promptly or death will ensue.



5A 5B

FIG. 5. Diagrammatic cross section representing mechanics of flail chest. A, inspiratory phase showing sinking in of unstable chest wall; admixture of inspired air and air from lung on damaged side being taken into lung on unaffected side; shifting of the mediastinum toward the unaffected side. B, expiratory phase showing pushing out of unstable chest wall; partial expiration of air from the lung on the unaffected side into the lung on the damaged side; shifting of the mediastinum toward the damaged side. These phenomena comprise: (1) paradoxic movement of the chest wall; (2) Pendelluft respiration; and (3) mediastinal flutter.

Hemorrhage occurs from pulmonic or chest wall vessels. Hemorrhage from the lung itself may be exsanguinating if large pulmonary vessels are opened, and occasionally operation with control or lobectomy or pneumonectomy may be necessary. Most often, however, blood loss from the lung is not great enough in itself to cause death from hemorrhage and it usually controls itself. Low pressure in the pulmonary vessels and frequent partial collapse of the lung due to pneumothorax or hemothorax assist in hemostasis. With traumatic rupture of bronchial or alveolar structures there may be rupture of blood vessels with intrabronchial, intra-alveolar or interstitial hemorrhage. This hemorrhage may seriously interfere with respiratory function by filling air passages and interstitial tissues with blood.

This is the mechanism of fatality following blast injuries to the lung. In these injuries there is extensive rupture of bronchioles, alveoli and their blood vessels, literally drowning the patient with hemorrhage and transudate.

Hemorrhage from the thoracic wall, i.e., intercostal and internal mammary vessels, is a frequent problem and exsanguinating hemorrhage may occur from either of these sites. In a few cases of injury, moderate to severe hemothorax manifests itself after a three- to eight-day period of freedom from clinical signs and symptoms (late hemothorax). The intercostal arteries, except for the first and second, come directly off the aorta, are of good size posteriorly and carry high pressure. An injury, particularly posteriorly, to an intercostal vessel may have to be controlled directly. This may be a treacherous injury because the bleeding, like that of the internal mammary, may be within the chest and concealed. The internal mammary vessels are also of large size, come off the subclavian arteries and carry high pressures. The bleeding from these vessels is most often concealed within the chest, but their location, about 1 cm. lateral to the sternal

border on each side, will lead to a high index of suspicion of injury to this vessel in all wounds in this area.

In controlling hemorrhage from the intercostal and internal mammary vessels, the collateral arterial channels in each case must be remembered. (Fig. 6.) Because the internal mammary arteries connect with the intercostals (to the fifth anteriorly), musculophrenic and superior epigastrics, these vessels have a potent arterial anastomosis peripherally and unless both sides of any bleeding point are ligated, bleeding will not be controlled.

In our experience control of the intercostals is accomplished chiefly by proximal ligation. Their distal ends, although anastomosing through connecting branches with the internal mammary in the upper five vessels, are of small caliber and do not produce a problem of retrograde bleeding. (Fig. 6.)

Pleural shock is a state of circulatory depression which may be incident to penetration of the pleural membrane or the sudden development of pneumo- or hemothorax. It appears to be a primary or reflex phenomenon and is seldom severe or prolonged.

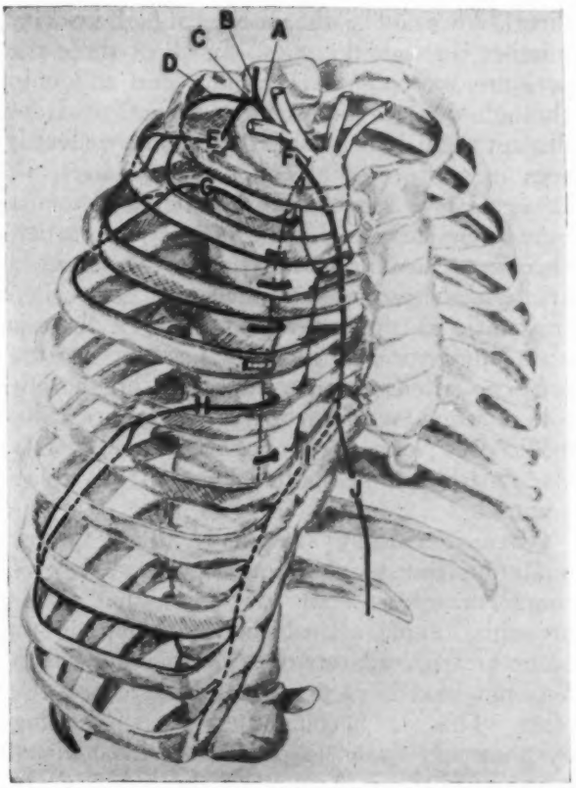


FIG. 6. Diagram showing the origins of the intercostal, intercostal collateral and internal mammary arteries on the right side, the courses and relationships of these vessels to the thoracic cage and their major anastomotic connections. A, costocervical trunk; B, deep cervical branch; C, superior intercostal; D, first intercostal; E, second intercostal; F, internal mammary; G, third intercostal; H, eighth intercostal; I, musculophrenic; J, superior epigastric.

SPECIFIC PULMONARY INJURIES AND THEIR TREATMENT

The various pulmonary injuries and their treatment may be analyzed in terms of the development of a number of morbid components associated with these injuries. These morbid components are: (1) Shock, (2) anoxia, (3) hemorrhage, (4) pneumothorax, (5) the unstable chest wall and (6) infection.

Emphasis on the aforementioned components will vary according to the type and extent of the injury.

An outline of the principal lung injuries follows:

I. Non-penetrating injuries to lung
   - A. Blast injury
   - B. Burns
     1. Chest wall burns
     2. Inhalation burns
   - C. Inhalation trauma
   - D. Radiation trauma
   - E. Crushing trauma to chest

II. Penetrating injuries to lung
   - A. Foreign bodies
   - B. Rib fractures

NON-PENETRATING INJURIES TO THE LUNG

Blast Injury. Blast or concussion injury is caused by the transmission of shock or pressure waves through the chest of a victim who is in proximity to the point of detonation of an explosion. The severity of the injury is related to the nearness of the victim to the blast, the type of explosive (high explosives cause more damage) and the medium through which the pressure wave is transmitted before reaching the body. Shock waves passing through water are much more damaging than waves originating from a similar explosion in air. This is because water is heavier and much less compressible than air. This allows it to transmit the shock wave faster and with much more sustained intensity from its point of origin. The same pressure phenomenon is caused by localized contusions of the chest following falls or

direct blows and by the passage of high velocity missiles through the chest. In all of these the pressure wave, passing rapidly and suddenly through delicate pulmonary tissues, tends to disrupt them. The principal pulmonary effect is seen in rupture of alveoli and capillaries, although larger bronchial and vascular elements may be similarly damaged. As described earlier, there is an ensuing permeation of the damaged area with transudate and blood and a so-called traumatic wet lung develops. Grossly, there is a transformation of the normal spongy aerated organ to a dense, airless, hepatized organ. The role of reflex factors in the production of traumatic wet lung have not been completely elucidated, although they undoubtedly play a part.

After a patient sustains a blast injury numerous moist rales and a wet, ineffective cough develops. With the increasing intrabronchial fluid, atelectasis and pneumonitis occur and spread to areas of the lung which may not have been primarily damaged by the blast. This is accompanied by increasing dyspnea and cyanosis. When the blast effect is generalized, as in some of the more severe blast injuries, treatment is ineffective and death ensues promptly. In the more localized or less severe cases the use of measures to combat anoxia, atelectasis and infection may be successful. Anoxia in these patients is treated by encouraging and assisting the patient to cough up endobronchial fluid. Close supervision with good nursing care, minimal sedation, particularly the avoidance of narcotics that may depress the respiratory center in therapeutic dosage, the administration of oxygen and the use of intratracheal suction, bronchoscopy and occasionally tracheotomy to remove secretions are the methods of approach. With effective removal of secretions and transudate, the atelectasis is prevented or relieved.

Adequate doses of antibiotics must be given to help prevent and control pneumonitis. General supportive care is given, but the use of infusions and transfusions must be undertaken with the utmost care. Electrolyte solutions, especially, aggravate the condition of wet lung. In the management of these cases one must appreciate the amount of local lung damage and this must be weighed against the systemic fluid imbalance that exists, remaining fully aware that the injudicious use of electrolyte solutions constitutes a definite hazard. When the patient's condition or other associated injuries dictate the use of supportive fluids, whole blood, slowly administered under close supervision, has been our preference.

Burns. Thermal burns applied to the chest wall seldom penetrate deeply enough to burn the lung itself, except in cases of advanced charring and destruction of the chest wall by flame, in which case the patient seldom survives beyond rescue. Burns of the chest wall do exert noxious effects upon the lung, however, by restricting motion of the chest in two ways. Painful first and second degree burns of the chest wall cause marked guarding and splinting of the chest. This is a conscious as well as a reflex attempt to avoid motion of the chest which would increase the pain. Also, third degree burns, although not painful, may present a dense, inelastic, leathery, coagulation en cuirasse which will mechanically restrict the respiratory motion of the chest. Abdominal breathing will also be restricted by these same mechanisms operating in the abdominal wall. The total effect on the lung is the same. Restricted motion makes it difficult to rid the tracheobronchial tree of secretions. Retained secretions produce blockage and atelectases, anoxia and pneumonitis. Reflex mechanisms, demonstrated experimentally by Detakats[8] in a wide variety of chest wall injuries, may also operate here to increase tracheobronchial mucorrhea and hasten the process of atelectasis. It is apparent, therefore, that the effect of burns of the chest wall on the lung are independent of the direct thermal effect on the lung. However, this direct effect may occur to a relatively minor degree in some cases.

When flame, or hot air or gases are inhaled by the patient, burns are produced through the respiratory passages, from the external nares down through the oropharynx, glottis, larynx and tracheobronchial tree. Depending upon the intensity of the heat and the amount of inhalation, the burns may extend to or past any of the structures named, and they may be involved in first, second or third degree burns. The events which follow are: swelling of the involved mucous membrane and hypersecretion by glandular elements, infection of the damaged mucous membrane and the addition of suppurative material to the glandular mucorrhea. Swelling of the laryngeal and glottic areas prevents effective coughing and also produces respiratory obstruction and stridor. This

necrotizing tracheobronchitis and respiratory obstruction soon produce bronchopneumonia which kills the patient.

Burns of the nasal passages and pharynx carry a very grave prognosis even though they may be accompanied by only a small burned area of the face. When first seen the patient may appear to be comfortable and stabilizing well following his injury. The presence of hoarseness and a cough is the indication that the burn has extended into the respiratory tract. When the inevitable processes of glottic edema and suppuration within the tracheobronchial tree supervene, and they may take from six to seventy-two hours, the mortality in these cases is extremely high.

Treatment in cases of burns of the chest, and particularly respiratory tract burns, must be concerned with the early and continued maintenance of a patent airway and clearing this airway of tracheobronchial secretions. Oxygen administration, catheter suction of the pharynx and tracheal suction, bronchoscopy and tracheotomy as indicated may be used. Large quantities of antibiotics are used to combat infection. Hygienic care of the oropharynx is maintained by suction and frequent irrigation with hydrogen peroxide and antibiotic-containing solutions. The too liberal use of intravenous fluids must be guarded against and oversedation with barbiturates or narcotics must also be avoided in the management of these cases.

Inbalation Trauma. There are a host of substances which when inhaled may traumatize the tracheobronchial tree and lungs by their chemical properties. Ficklen,[9] in his manual, reviews more than ninety vapors, gases and dusts which are important as industrial health hazards. In general, these substances act acutely as local irritants on the lung and tracheobronchial tree, or slowly as the pneumoconioses, producing pulmonary interstitial fibroplasia. There is also the group of war gases, which when inhaled give an intense irritative effect. Space will not permit a discussion of these individual substances.

By far the most common offender to which most of the population in urban areas is exposed is smoke. Smoke originates in domestic and industrial sources and may constitute a public health problem as well as an economic one by its irritative and destructive properties. The usual smoke which comes from the burning of coal contains varying concentrations of tars, grit, ash, sulfur dioxide and other chemicals. The sulfur dioxide combines with oxygen of the air and moisture to form sulphuric acid which has a destructive effect on clothing, metal, stone and other building materials and vegetation, as well as the respiratory tracts of persons breathing such fumes. The grit and ash also contain small amounts of silica which, after a time, will produce fibroplastic changes in the lungs of persons heavily exposed. Under certain atmospheric conditions, when smoke in a given area is not diluted and dispersed, a condition of concentrated smog or smaze may develop and continue for days at a time. Pulmonary irritative phenomena may then constitute a public health problem of major proportions. Pharyngitis, laryngitis and tracheobronchitis with mucorrhea and cough develop in persons exposed. During these periods the death rates from respiratory diseases and heart diseases rise significantly as a direct result of superimposition of this pulmonary effect.

Radiation Trauma. Radiation trauma to the lung is a class of injury which until recently has been seen relatively rarely and usually in relation to the use of radiation therapy to the breasts. Our experience with this type of injury on the surgical service at Harlem Hospital has been minimal. Now, with the increasing use of radioactive materials and particularly the possibility of the use of thermonuclear weapons in warfare, we may expect to see this injury more frequently.

A working concept of the pathology of this type of injury differs from that of a thermal burn in the following ways: First, it occurs in depth, depending upon the penetrability of the given radiation. Second, there are variable latent periods following the exposure before the symptoms of injury develop. Third, the effects are likely to be more severe and prolonged than a thermal burn, although the last two characteristics are dependent upon the type and duration of the radiation. Hematopoietic tissue particularly is severely damaged by penetrating radiation.

The details of the tissue changes resulting from ionizing radiations are still very poorly understood. It appears that damage, in a large part, is due to excess of oxidative reactions in the cells, induced by oxidizing radicals which are liberated by the radiation-induced dissocia-

FIG. 7. Photograph demonstrating tracheobronchial suction. Assistant holds tongue forward and directs light into pharynx. Operator introduces a stiff rubber tube through nostril into oropharynx. Suction is then started while tube is being introduced into trachea. The open mouth proves of value in directing the tube.

tion of water and other tissue components. This concept forms the rationale for administering reducing substances containing thiolic (—SH) or aminic ($—NH_2$) groups to protect against the tissue effects of radiation.

The tissue reaction to radiation in lung begins with hyperemia, edema and round cell infiltration and progresses on to bleb formation and varying degrees of cellular necrosis, followed by infection and suppuration. This process in the lung bears an obvious relationship to the development of respiratory complications. There have been few accurate and detailed studies of radiation pneumonitis, although the course of events, as indicated by the work of Lobsenz,[10] makes it appear that in marginal and sublethal injuries non-specific treatment of the resulting pneumonitis with antibiotics, oxygen administration and the maintenance of an adequate airway may be expected to have good effect. The use of cysteinamine or other reducing substances, vitamin B_{12}, and hematinic compounds and antibiotics may be of value in ameliorating the systemic effects of exposure to radiation. Work is continuing in this field but at the present time there appears to be no substance as yet available which will give significant protection against, or arrest and reverse, the cytologic effects of intense radiation exposure such as would be produced by an atom or hydrogen bomb.

Crushing Trauma to the Chest. Crushing injuries to the chest occur frequently in falls, automobile and airplane accidents, and other forms of gross physical trauma to the body. Their immediate mortality is high because they are frequently associated with rupture of the heart or great vessels, fatal spinal and cerebral injuries, extensive fractures and decapitation or other dismemberment of the body. Of itself, however, a crushing injury to the chest may be rapidly fatal by setting into motion the chain of events which have been described earlier in this paper. Any part of the chest may be stove-in and a particularly lethal situation arises when both chests are crushed or when both chests are made unstable by extensive bilateral costochondral fractures and a flail sternum. There may or may not be a penetration into the chest and pneumothorax, but the mechanical forces previously described produce anoxia, shock and death.

Treatment is primarily directed at stabilizing the chest wall. This may be done by sandbags or strapping if the area is unilateral and fairly small in size. More extensive instability must be handled by traction to the unstable sternum or chest wall by tenacula, towel clips or wires. Direct wiring or bone grafting of the unstable ribs can be done in some instances. However it is done, the stabilization of the chest wall is mandatory and ingenuity is sometimes required to accomplish this. No one method of stabilization is applicable to all cases. The maintenance of chest wall stability may be assisted by the use of positive pressure oxygen, either by mask or by tracheotomy tube. Jansen's method of keeping the pressure of the air in the tracheobronchial tree always somewhat above atmospheric pressure, even during expiration, has been reported as reducing paradoxic movement of the unstable chest and flutter of the mediastinum.[11] At the same time this is effective treatment for the pulmonary edema or wet lung. We have not committed ourselves to a use of this method because of danger in impeding expiration with positive pressure and the lack of a well standardized, mechanical breathing apparatus.

An open and clear airway must also be maintained. Frequently this can be accomplished by transnasal suction. (Fig. 7.) Tracheotomy, in order to avoid obstruction at the

laryngeal or glottic level and to enable tracheobronchial secretions to be removed easily and repeatedly, should be performed without delay if it appears needed. Supportive treatment in the form of shock therapy, antibiotics, judicious sedation and the care of associated injuries completes the plan of therapy.

Pneumothorax and hemothorax also frequently occur following multiple rib fractures in this type of injury. The handling of these complications is discussed in the following section.

PENETRATING WOUNDS OF THE LUNG

Penetrating trauma to the lung may be inflicted by foreign bodies such as knives, bullets and other missiles or by the fragment of a broken rib, where displacement so as to impinge upon the lung occurs.

Foreign bodies which are of low velocity, such as knives, and the penetration of rib fragments cause localized tissue damage in the site or tract of injury, but in general such wounds are tolerated well by the lung and unless infection supervenes, healing is prompt. Furthermore, bleeding is not likely to occur in massive amount from the lung, and the leakage of air into the pleura is only temporary, the defect sealing itself promptly as a rule.

With penetration by high velocity missiles, such as bullets and shrapnel, the tissue defect is likely to be larger and this defect is surrounded by a zone of devitalization due to blast or concussive effect. Therefore, the complications of pneumothorax, hemothorax and infection are more frequent and severe. Surgery in these cases is usually confined to the débridement and care of the external wounds. Further surgical operative management will be required when bleeding from lung, intercostal artery or internal mammary artery are not controlled, or when lung or thoracic wall damage is very extensive. Exploration may also be indicated to rule out or repair damage to heart, great vessels, trachea, bronchi or esophagus. Exploration will also be indicated in cases of diaphragmatic injury and in this instance abdominal exploration as well may have to be performed. The extent of pneumothorax may be followed by watching the physical signs (auscultatory signs, respirations, shock, etc.), performing serial pneumothorax readings and taking x-rays of the chest. A large or sucking wound of the chest must be closed immediately. One must be on the alert for progressive pneumothorax or the development of tension. A stabilized pneumothorax, even of fairly large degree, may do no harm if the patient tolerates it well and it may have the advantage that the partially collapsed lung tends to control air leaks and hemorrhage quicker. Therefore, we believe that if pneumothorax is minimal or moderate and not increasing, and the general condition of the patient is stabilized, no urgent evacuation of the pneumothorax is necessary. A persisting pneumothorax may be removed later at a convenient time by aspiration.

If the pneumothorax is massive when seen or is increasing in size, or if there are signs of encroachment upon respiratory function, needle aspiration or decompression by underwater drainage must be performed immediately. If there is evidence of increase, persistence or recurrence of the pneumothorax so treated, a small rubber tube placed into the chest through a trocar and led to underwater drainage, or a polyethylene tube similarly placed through a large bore needle (15 gauge) provides sure and continuous decompression of the pleural cavity. Only relatively rarely will it be necessary to explore a chest to control air leaks from lung or bronchi.

With hemothorax it must be remembered that dangerous hemorrhage occurs most often from the thoracic wall rather than from the lung. The factors that govern the decision to operate to control hemorrhage are location of the wound (areas of internal mammary and posterior intercostals, especially), size of the hemothorax when first seen and its subsequent progress, and other evidences of continuing or massive hemorrhage such as falling hemoglobin and hematocrit, pallor, air hunger and shock. Chest wall vessels, internal mammary and intercostal, may be approached and ligated extrapleurally. However, it must be remembered that massive hemothorax may follow injuries to the heart, aorta, vena cavae, or major pulmonary vessels, and open thoracotomy will be dictated when there is a suspicion of injury to any of these structures. We have had four cases in which there were combined injuries of the vessels of the internal mammary, and the heart.

If the hemothorax is small or moderate, close observation of the patient and x-ray and auscultatory checks of the extent of the hemo-

thorax must be made. If distress occurs due to pressure of the hemothorax or mediastinal shift, immediate aspiration is performed to give relief.

A hemothorax should be evacuated by repeated needle aspiration to remove as much of the blood as possible. The sooner that this is done after cessation of bleeding, the greater the likelihood of success in complete evacuation. Instillations of streptokinase may aid in this, although there have been reactions encountered with the use of the drug and it may predispose to a recurrence of bleeding. We favor beginning aspirations within twelve to twenty-four hours after cessation of hemorrhage and stabilization of the patient. Daily aspirations are performed until no more blood can be obtained.

The sequel to an incompletely drained hemothorax is a captive, hypofunctioning lung enclosed by organizing blood clot which must be removed surgically by decortication. Small degrees of residual clotted hemothorax may be absorbed. It is our procedure, however, to perform early decortication after three to six weeks if there is not continued significant diminution in the pleural clouding on follow-up x-rays. Decortication at this time is technically easier and the continuing functional capacity of the lung is assured.

Rib Fractures. Fractures of ribs, where only one or two ribs are involved and there is no threat to the stability of the chest, may be best treated by intercostal block with xylocaine or a similar local anesthetic to relieve pain. Strapping of the involved area of the chest with adhesive tape or elastoplast bandage also gives relief from pain at the expense, however, of diminishing the movement of the thorax. This may have a distinct disadvantage in an older patient, especially when a wide segment of the thorax is involved. Direct wiring for fixation of ribs or a combination of multiple intercostal blocks with gentle taping may be recommended in more extensive cases. The unstable chest in multiple rib injuries has been discussed.

SUMMARY

A brief consideration has been made of the pathology of the principal types of lung trauma with incident or closely allied trauma to chest wall and pleura.

It has been emphasized that failure of respiratory function secondary to a number of mechanisms is the lethal factor in most of these injuries.

Specific injuries are discussed and outlines of therapy given, based largely upon our experience in the management of cases at Harlem Hospital.

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Pleuropulmonary Resection

EXCISION OF CHRONIC EMPYEMA

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PLEURAL involvement is a frequent complication of pulmonary malignancies, suppuration or tuberculosis. Until quite recently pulmonary resection was often unfeasible because the surgeon could not overcome the serious technical problems occasioned by the presence of dense and widespread pleuropulmonary adhesions (pleural symphysis) or empyema.

In tuberculosis particularly, and in chronic pulmonary suppurations the difficulties in resection caused by the presence of pleural symphysis served for many years to keep pulmonary extirpation from achieving its present acceptance as a form of therapy. Not infrequently operations which had been begun to remove a lung had to be abandoned because the lung could not be separated from the chest wall; or the operations were so prolonged by dissection through dense adhesions and manipulation of the lung so great that operative shock was frequent, anesthetic difficulties due to spillage of secretions common and postoperative spreads of disease not unusual. Rupture of adherent cavity walls and incision of diseased parenchyma or pleura were frequent sources of contamination.

A second limiting factor was the tenet that pleural infection or empyema was a contraindication to pulmonary resection.[1] Many patients with pulmonary disease suitable for such treatment were denied resection because empyema was present. In other cases excisional procedures already begun were not completed because of the unexpected operative finding of an empyema.

Although Tuffier[30] as early as 1913 had successfully resected the tuberculous apex of a lung extrapleurally, and Fowler[9] in 1901 had noted the importance of removing the entire diseased pleura in empyema, these isolated observations were ignored because of a universal reluctance to damage or remove the parietal pleura surgically. This reluctance was based on the accepted belief[11] that under no circumstances should the parietal pleura be removed, especially in tuberculosis, since its removal would permit the spread of infection to tissues beyond.

THE EXTRAPLEURAL APPROACH[22,23]

Extrapleural dissection in lobectomy and pneumonectomy has made it possible to circumvent the difficulties occasioned by dense and widespread adherence of the lung and to excise an empyema with the diseased part of the lung. Extrapleural dissection is of particular value in approaching the hilus. When the hilar area is exposed extrapleurally through a posterior approach (Fig. 1), the thickened pleura with its adhesions is bypassed and the hilar structures are exposed in a region relatively uninvolved by disease. The connective tissues about the bronchus and the pulmonary vessels in the mediastinum are not usually involved to any great degree by the inflammatory processes of the lung and pleura.

The extrapleural approach to the hilus for pneumonectomy avoids difficult dissection through matted nodes since the greatest degree of lymph node involvement in inflammatory states is usually at the bifurcation of the main bronchus, distal to the region in which the bronchus is exposed. The infratracheal nodes and the peribronchial nodes of the main bronchus usually separate from the bronchus quite easily if the peribronchial connective tissue sheath is incised and the bronchus freed within it. The division of the bronchus at the carina, the suturing of the proximal end and the ligation of the vessels are achieved in an unobstructed and clean field. The lung with the adherent pleura, or if empyema is present, with the entire empyema sac, is then freed by extrapleural dissection and removed.

The widest application of the technic of extrapleural resection has been in pulmonary tuberculosis[22] in which cases with complete pleural symphysis or empyema, previously

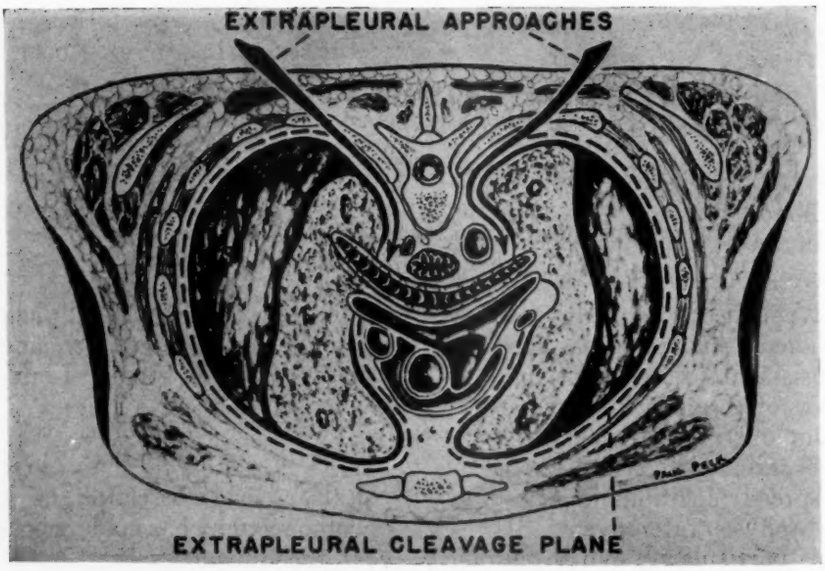


FIG. 1. Diagrammatic sketch illustrating the posterior extrapleural approach. (By courtesy of the Editors of *Thorax* and the British Medical Association.[22])

considered inoperable, have become suitable for resection. (Fig. 2.) Tuberculous or mixed infection empyema with or without bronchopleural fistula, previously a most difficult condition to control or cure, especially when complicated by underlying active pulmonary tuberculosis, can now be treated successfully by extrapleural pulmonary resection and pleurectomy with a mortality and morbidity rate no greater than that in resection for extensive but uncomplicated pulmonary tuberculosis.[25] Extrapleural resection has thus become important in the surgical treatment of pulmonary tuberculosis, extending the indications for resection, lessening complications and improving results.

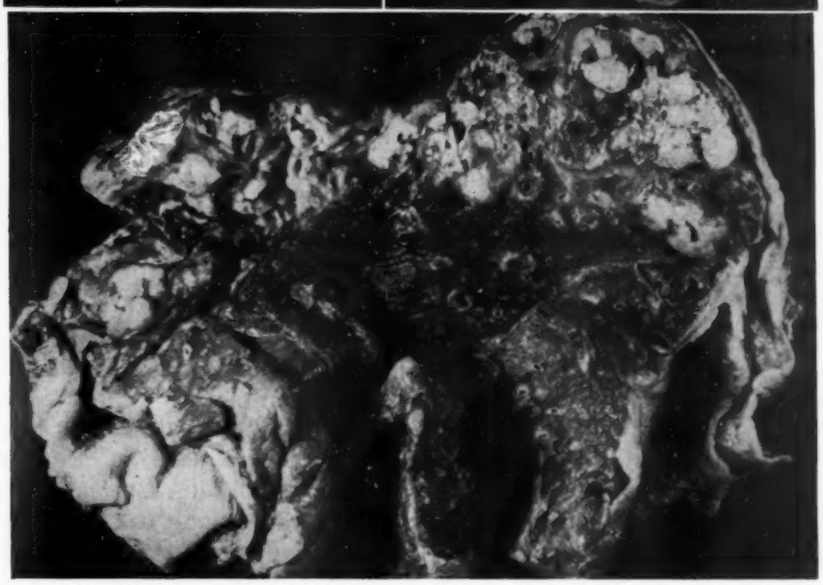
Extrapleural resection is applicable to nontuberculous disease.[23] Many surgeons have reported the freeing of very adherent portions of the lung by limited extrapleural dissection as a matter of convenience at the moment. However, it has not been recognized generally that this should be the procedure of choice. When the extrapleural procedure is planned and developed primarily, tedious and often dangerous intrapleural dissection of adherent lung is avoided. Since, as already mentioned, the mediastinal aspect of the lung and the mediastinal pleura are relatively uninvolved in extensive inflammatory processes of the lung and costal and diaphragmatic pleura, it is usually possible to approach the hilus extrapleurally with ease even in cases in which the entire lung is destroyed by chronic abscesses or bronchiectasis. This permits the bronchus to be clamped early in the operative procedure, lessening the danger of bronchial dissemination of infected secretions. After the hilar structures are divided, the lung can be freed with less danger of hemorrhage.

Circumscribed carcinomas of the lung may be of the "parenchymal" type entirely within the substance of the lung, or of the "peripheral invasive" type which may actively invade the adjacent chest wall, diaphragm or mediastinum.[16] The former type usually has a pseudocapsule about it, is not invasive locally, but may, when it involves the surface of the lung, be densely adherent to the regional parietal pleura. Such a tumor may be resected conveniently with the adherent pleura by extrapleural resection. When circumscribed carcinoma of the peripherally invasive type involves parietes which are resectable, the involved chest wall and adherent and adjacent pleura must be resected *en bloc* with the neoplasm and lung. If, during the surgical exploration of a case of carcinoma of the lung, the lung is found to be densely adherent to the parietal pleura, it may be freed and the hilar structures exposed more safely and easily by extrapleural dissection than by attempting to separate the lung from the parietal pleura.

Metastatic pleural involvement from underlying pulmonary carcinoma occurs frequently, and the presence of pleural effusion containing

2A

2B



2C

FIG. 2. Lung destroyed by tuberculosis with bronchopleural fistula and empyema; pleuropneumonectomy. A, lateral view; empyema has been opened to demonstrate bronchopleural fistula; B, mediastinal aspect shows characteristic sharp delineation of pleural thickening and relative uninvolvement of mediastinal pleura; C, cut surface.

carcinoma cells is considered a contraindication to resection by most surgeons. However, in suitable cases the pleura and lung may be removed as a palliative measure. Even when the diaphragm is intrinsically involved, the reaccumulation of fluid in the thoracic cavity abates when the parietal pleura has been removed and, aided by the addition of cobalt or radioactive gold therapy, some palliation may be obtained. Cotton[4] has added hemidiaphragmectomy in such cases. He has also reported successful pleuropneumonectomy in pleuropulmonary involvement by higher bacteria.

THE ROLE OF THE PLEURA

As the scope of application of pleuropulmonary resection has been broadened in the treatment of pulmonary and pleural inflammatory and neoplastic processes, it has been inevitable

that concepts and ideas previously accepted should be re-examined.

Tchertkoff and Selikoff[29] first pointed out that extrapleural resection which began as a technical convenience achieved a greater effect by removing the parietal pleura. This membrane has no useful function after pneumonectomy; its removal eliminates its great reactive and secretory power and any residual foci of pleural infection from which empyema may develop if the pleura is permitted to remain. The pleura is sensitive to infection, responding to contamination with exudation and in tuberculosis with caseation and empyema. In contrast, the extrapleural tissues which line the thoracic cavity after the parietal pleura has been removed are relatively resistant to infection and respond to contamination with productive changes rather than empyema. Contamination of the operative field from rupture into a tuberculous cavity or from incision through densely adherent tuberculous tissue or suppurating lung is largely eliminated in extrapleural dissection. Rupture into an empyema (when present) occurs frequently but such contamination is less likely to be followed by empyema after the pleura has been removed.

When thoracostomy and cavernostomy openings and empyema necessitatis and other sinus tracts are transected during intrapleural pneumonectomy, empyema or wound infection is invited despite antibiotics. In contrast, when they are removed *en bloc* during pleuropulmonary resection, empyema and prolonged wound infection are less likely to result.

Bronchopleural fistula is still the most dangerous postoperative complication following pneumonectomy. Other than residual malignant disease and tuberculous or other intrinsic disease in the stump of the resected bronchus, the two factors which seem to act to cause such fistulas are postoperative local infection or the presence of a long bronchial stump which is prevented from retracting into the mediastinum by adhesions to the pleura and to the extrapleural connective tissues.[24] It is probable that the lesser incidence of clinically evident bronchopleural fistula following extrapleural dissections is due in part to the reduced incidence of infection and empyema after pleurectomy and also to the greater ease with which a high bronchial amputation close to the carina can be obtained. A short bronchial stump freed from the surrounding hilar and extrapleural fascia retracts into the mediastinum and is covered promptly by the adjacent tissues. Stemmermann, Daniels and Auerbach[28] have recently stressed the role of local infection in the pathogenesis of postpneumonectomy bronchial fistula.

During an extrapleural dissection one may encounter extensive fibrosis in the extrapleural apical tissues and must guard against damage to the large vessels. Policard and Galy[18] have studied the many layers in the extrapleural fascia and have demonstrated well our operative finding that one does not always dissect in the same cleavage layer. In fact on occasion it is necessary to dissect extrafascially when dense inflammatory reaction has sealed the extrapleural tissue planes. Great difficulty may arise during the dissection from the inferior vena cava on the right side and during the dissection from the diaphragm. Usually the hilar structures are severed and the apical and mediastinal dissection completed before the diaphragmatic dissection is begun. The cleavage plane between an empyema wall, or the diseased lung and the diaphragm is often difficult to enter. Policard and Galy[18] and others have shown that the diaphragmatic pleura differs from the other portions of the parietal pleura in that the deep fibroelastic layer is intimately connected with the muscle of the diaphragm and must be considered part of it. For this reason a cleavage plane may be difficult to obtain, especially in diseased states. A cleavage plane can be developed and the diaphragm identified more easily after it has been paralyzed by interruption of the phrenic nerve. The diaphragmatic cleavage plane can always be entered more easily from the mediastinal aspect where there is less pleural involvement than if dissection is begun in the costophrenic sulcus where the adhesions are usually more severe.

PLEUROPULMONARY RESECTION AFTER THORACOPLASTY FAILURE

After thoracoplasty (and possibly Schede resection) has failed in the treatment of empyema associated with perforated pulmonary abscess, bronchiectasis or tuberculosis because symptoms persist due to the underlying chronic pulmonary disease, extrapleural dissection makes it possible to remove all or part of the lung when an intrapleural dissection is impos-

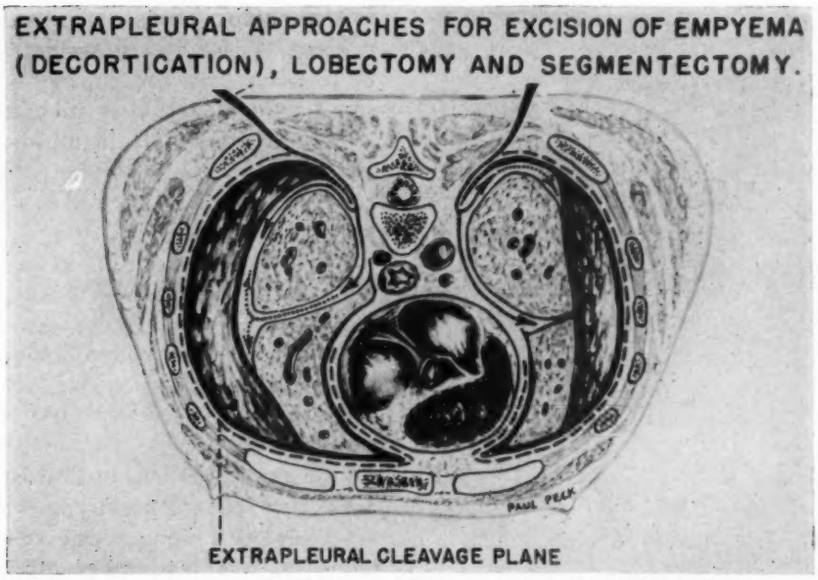

EXTRAPLEURAL APPROACHES FOR EXCISION OF EMPYEMA
(DECORTICATION), LOBECTOMY AND SEGMENTECTOMY.
EXTRAPLEURAL CLEAVAGE PLANE

FIG. 3. By courtesy of the Editors of *Thorax* and the British Medical Association.[22]

sible technically or fraught with the danger of infection due to operative rupture of the diseased lung or incomplete removal of diseased pleura and lung.

In tuberculosis the results of treatment of residual cavities by revision operations have been disappointing.[10,15] The success achieved by resection of the diseased lung in these cases is particularly gratifying. The antecedent thoracoplasty may render the extrapleural excision more difficult technically, but the postoperative course of these cases is notably benign.

Usually the scar of the previous thoracoplasty is excised, the uppermost intact rib below the thoracoplasty is resected paravertebrally and the extrapleural plane entered through its bed. The regenerated bony plate above is cut paravertebrally and lifted away by a rib spreader as the underlying lung is stripped from it extrapleurally, or even extrafascially to lessen the risk of opening into a cavity or diseased lung.

The extrapleural dissection is continued to the hilus in the usual manner and the main bronchus and vessels are divided if the preoperative indication has been definitely for pneumonectomy. If the extent of the disease permits, partial excision of the lung is possible. The mediastinal (parietal) pleura which has been freed is incised and the proper cleavage plane between parietal and visceral pleura is entered. In tuberculosis usually, and in other inflammatory conditions frequently, the mediastinal regions of the interlobar fissures are less involved in the inflammatory processes and adhesions less pronounced. Therefore these fissures may be entered here more easily and the lobes separated. Lobectomy and even segmental resection are facilitated. (Fig. 3.) Densely adherent upper lobes, especially those containing large cavities close to the surface, are freed more easily if the dissection is done extrapleurally. The densely adherent pleura should be removed with the lobe or segment, but the less adherent and adjacent pleura may be permitted to remain after lobectomy or segmentectomy to exercise its absorptive and gliding functions to aid in the prompt reexpansion of the remaining lung. The lobar bronchus and vessels may be dissected through the extrapleural hilar approach. Division of the azygos vein extrapleurally on the right side aids in exposing the right upper lobe bronchus and the apical branch of the pulmonary artery.

The average intrapleural pneumonectomy requires about 2,000 to 2,500 cc. of blood during and immediately after operation. A difficult extrapleural resection may require two to three times as much, but this disadvantage of greater blood loss is more than compensated for by the other advantages of the extrapleural approach.

PLEUROPULMONARY RESECTION FOR CHRONIC PULMONARY DISEASE WITH EMPYEMA

The beneficial effects of isoniazid in tuberculosis (in combination with streptomycin and

FIG. 4. Tuberculous right upper lobe resected extrapleurally with chronic tuberculous empyema.

PAS) have been demonstrated universally since its introduction into clinical use in 1952. Because of this new chemotherapeutic agent many formerly accepted concepts of treatment are in the process of revision and surgery is being withheld, perhaps wrongly, in some patients with pulmonary cavities but persistently negative sputum. Only further time and study will clarify this problem.

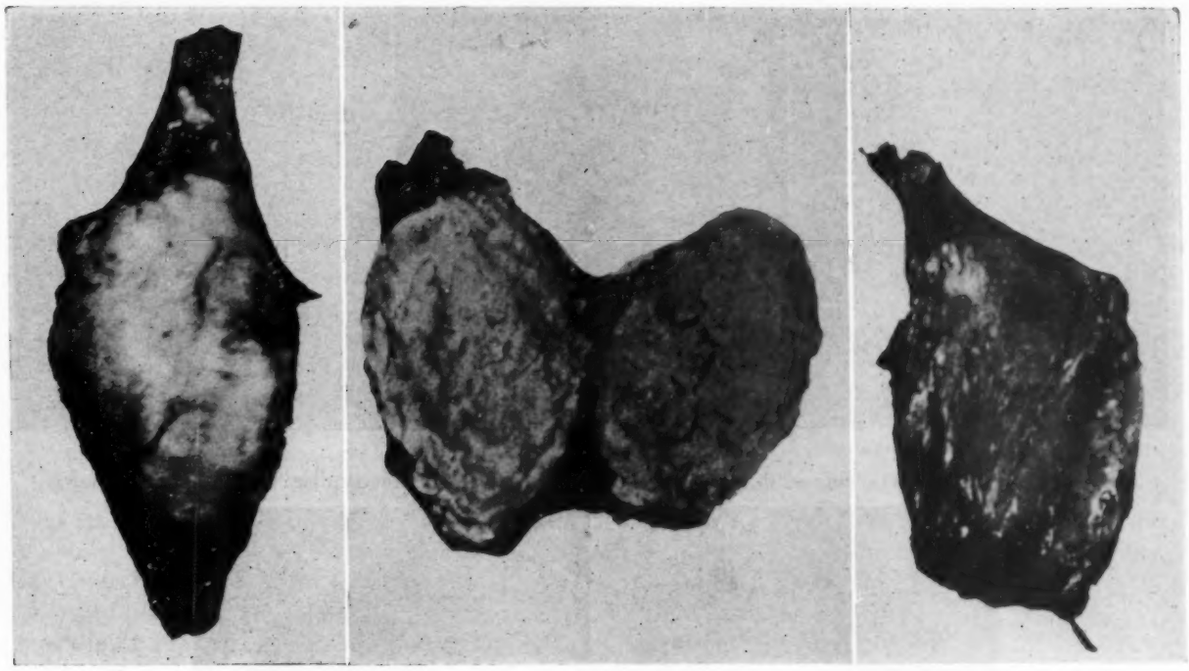
The concomitant decline in pneumothorax therapy has acted to make empyema a less frequent and pressing problem. Greater attention is again being given to the treatment of empyema by injection of chemotherapeutic agents and antibiotics and by repeated aspirations. Trypsin or varidase® controlled by an intercostal tube drain may facilitate rapid re-expansion of the lung in early empyema, especially if no bronchopleural fistula is present. If resection becomes necessary after the lung has been re-expanded, the pleura should be resected with the lung to avoid subsequent empyema from residual foci of pleural infection.

Siddons and Konstam[27] have recently reviewed a series of cases of tuberculous empyema (treated before isoniazid was available) and have reported the good results of careful and complete aspiration therapy. However, they stress the importance of prompt surgical intervention when a patent pulmonary cavity, bronchopleural fistula or unexpandable lung is present. These are the same indications which have been presented previously[21] for pleuropulmonary resection in such cases.

Spontaneous perforation of the lung in a patient not yet under chemotherapy for acute tuberculosis need no longer carry with it the lethal significance of former years, and early tube drainage may suffice to tide the patient over the acute phase of tension pneumothorax. The lung may re-expand rapidly to obliterate the pleural space while chemotherapy controls the disease and helps the fistula to heal. In some cases the patient may not even run a significantly febrile course and the external wound may heal without excision.

Despite chemotherapy, perforation secondary to badly managed pneumothorax or pneumonolysis still leads to empyema; spontaneous perforation does still occur, although less often, in patients under adequate chemotherapy, resulting in severe empyema with or without persistent bronchopleural fistula. The indication for prompt total or partial pulmonary resection and pleurectomy still exists in such cases to save lives and shorten the duration of the disease. Despite the extremely beneficial effect of chemotherapy, patients who are not doing well must not be permitted to wait too long before active surgical measures are applied, for if such cases become chronic despite intensive chemotherapy the outlook is worse.

Prior to the introduction of pleuropneumonectomy[19,20] the disappointing results obtained by all forms of surgical collapse therapy for uncontrolled pulmonary tuberculosis complicated by tuberculous empyema (with or without bronchopleural fistula) were part of the experience of every thoracic surgeon. One could not fail to be impressed, even in the successful cases, by the further permanent collapse of healthy lung and by the long period of surgical treatment that these patients suffered, yielding only meager results with much misery and deformity. Early mortality ran higher than 60 per cent in some series and cures were obtained in less than 15 per cent.[7] These results were the final products of drainage, subsequent complete thoracoplasty, Schede or Keller resections of the infected parietal (costal) pleura and, where necessary, plastic operations such as muscle-flap transplants to close the persistent bronchopleural fistula. The disaster which followed so fre-



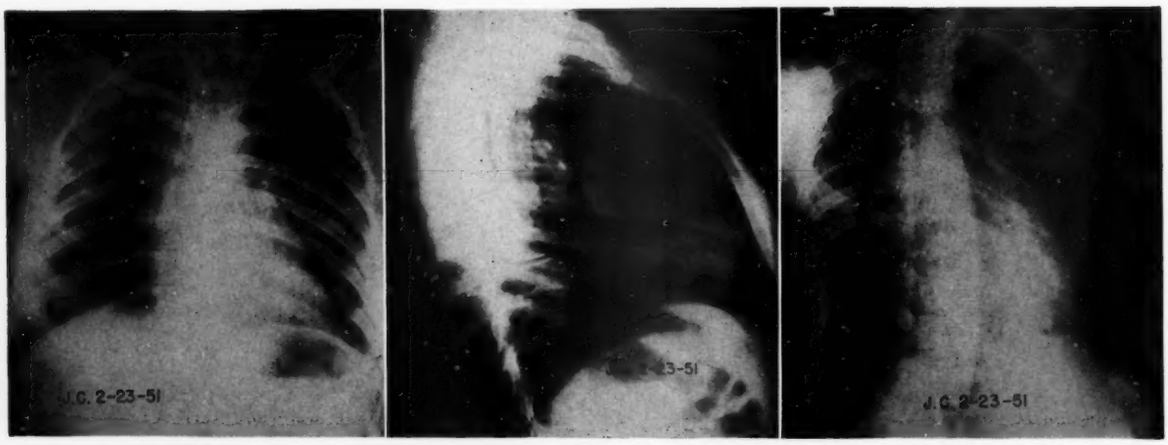
5A 5B 5C

FIG. 5. Excised empyema: A, visceral aspect; B, cut surface; C, parietal aspect. Tubercle bacilli were found in smear of pus but did not grow in culture or guinea pig.

quently was inherent in each case by virtue of the underlying disease which remained to contaminate the surgical field and to destroy the transplanted tissues. Even if the bronchopleural fistula healed, caseating infected pleura and visceral wall of empyema remained after Schede thoracoplasty and was a persistent cause of dissolution and ultimate non-healing of the wounds.

Analysis of all available experience with thoracoplasty in the treatment of uncontrolled pulmonary tuberculosis complicated by empyema indicates that the failure of this approach is due to several unavoidable deficiencies: (1) multiple operative procedures with increased opportunity for operative mortality; (2) prolonged duration of treatment inviting mortality from tuberculous spreads, toxemia and exhaustion, and amyloidosis; (3) failure to control the underlying lung disease; (4) the deleterious effect on wound healing of the residuum of infected caseating pleura; and (5) the frequent inability to control the bronchopleural fistula. Therefore any procedure designed to meet the problem of uncontrolled pulmonary tuberculosis and empyema must be (1) single stage, (2) of short over-all duration, and (3) must control the underlying lung disease, the infected caseous pleura and the bronchopleural fistula (if one is present). It is apparent that in order to cure a patient with uncontrolled pulmonary tuberculosis and a tuberculous or mixed infection empyema one must accomplish the removal *en masse* of the underlying diseased lung, the bronchopleural fistula (if one is present) and the entire empyema including the infected caseating parietal pleura. Pleuropneumonectomy fulfills these requisites. Its variations, pleurolobectomy (Fig. 4) and pleurosegmentectomy (partial pulmonary resection with excision of empyema), provide a means of excising the diseased lung and pleura while preserving and restoring to function the non-diseased parts of the lung in contrast to the usual effect of thoracoplasty.

In cases of limited involvement of the lung under an empyema the extent of the disease cannot always be determined accurately by even the best methods before operation. By the extrapleural procedure with entry through the incision in the mediastinal pleura into the interlobar fissures and the cleavage plane between visceral pleura and parietal pleura or empyema visceral wall, the lobes can be freed more easily and decortication is facilitated. (Fig. 3.) Lung tissue which is palpably free of



6A 6B 6C

FIG. 6A, B and C. Chronic empyema with no evidence of residual pulmonary disease; preoperative views.

6D 6E

FIG. 6D and E. Same case. Appearance of chest following excision of the empyema in Figure 5.

disease and which expands well when inflated by the anesthetist after being *easily* decorticated should be conserved. Its prosthetic value in filling the hemithorax outweighs any possible subsequent demonstration of reduced function by spirometry. A lobe or segment which is not readily decorticated or which will not expand easily is undoubtedly the seat of chronic fibrosis or disease continuous with the pleural involvement. In non-tuberculous conditions it may be safe to leave such a portion of lung with its adherent pleura while resecting other parts; however, in tuberculosis the surgeon must be prepared to resect such questionably diseased lung tissue. Overconservatism on the part of surgeons in the resection of such doubtful areas has probably been a major factor in some of the poor results from decortication. Decortication alone is not often advisable in tuberculosis except in unusual cases in which there is gross pleural thickening following effusion with little pulmonary disease.[8,21]

Occasional cases of encysted chronic empyema, tuberculous (Fig. 5) or non-tuberculous, are encountered with no demonstrable residual pulmonary disease. Such an empyema may be resected through an intercostal incision by extrapleural dissection beyond the limits of the thickened parietal pleura. Entry into the pleural space is gained by incising the parietal pleura beyond the area of thickening surrounding the empyema and the lung is separated from the internal wall of the empyema which is then removed, frequently intact. (Fig. 6.) Such a procedure is preferable to prolonged drainage and possibly multiple rib resection to close the space.

The results reported previously[25] of three years' experience with pleuropulmonary resec-

tion in tuberculosis with empyema indicated a reversal of the figures previously obtained by surgical collapse measures. Over 80 per cent of the patients were restored to health. Further statistics of our cases will be reported in the near future. Other surgeons[2–6,12–14,17,26,31] have reported similar good results in treatment of uncontrolled pulmonary tuberculosis with empyema by pleuropulmonary resection and have agreed that this procedure is usually to be preferred for such cases except in rare and special instances. Thoracoplasty may be indicated: (1) if in a case of pulmonary disease controlled by extensive previous thoracoplasty a basal empyema should occur, recur or persist, and the possibility of re-expanding the lung does not exist because the chest cage has been collapsed, or because of the extent of the previously active pulmonary disease re-expansion should be undesirable; (2) if a case presents a laterally placed single small cavity (less than 5 cm. diameter) in an adherent and expanded upper lobe without much pleural thickening over the upper lobe, a basal empyema without bronchopleural fistula, and nodular disease is present in the lower lobe to an extent which would preclude leaving it after upper lobectomy but which may heal after thoracoplasty.

Since the advent in the past two decades of the sulfa drugs, penicillin and other antibiotics, chronic pulmonary suppurations have become much less frequent. However, cases are seen of severe suppuration in the lung with empyema secondary to bronchial carcinoma or other obstructing lesions. Such patients suffer most from the fever and cachexia due to the infection. Palliation may be achieved in cases of malignancy, and cure in benign conditions, by pleuropulmonary resection. Other types of chronic lung abscess and bronchiectasis may be complicated by pleural infection and this difficult combination is usually best treated in the same manner. When, however, an older or debilitated individual has localized empyema secondary to mild or asymptomatic bronchiectasis, especially if this is widespread or bilateral and pulmonary or cardiac function is poor, drainage and thoracoplasty may be preferable even if permanent drainage openings persist in the scar.

SUMMARY

In cases of combined pleural and pulmonary disease it is usually not adequate to deal with the pleura without treating the underlying lung. The technic of extrapleural dissection makes it possible for both components to be treated simultaneously and permits the fulfillment of the surgical principle of complete excision of the disease in many types of cases in which it was previously not possible to accomplish this desirable effect. Not only is resection facilitated but also complications are minimized. This has been clearly shown in tuberculosis as well as in non-tuberculous disease.

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Present Concepts Relative to Autonomic Nerve Surgery in the Treatment of Pulmonary Disease

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INTEREST in regard to the possible application of surgery upon the autonomic nerve system in the treatment of pulmonary disease has been sporadic. Scattered reports concerning the use of sympathectomy in patients with pulmonary tuberculosis appear to constitute the earliest attempts at application of pulmonary autonomic nerve surgery upon humans. No significant results were obtained, and interest lagged until the work of Kümmel[1] in 1923, when he described the use of sympathectomy in the treatment of bronchial asthma. In 1928 Leriche and Fontaine[2] described their experiences with a similar approach to the surgical treatment of the same disease. In 1938 Reinhoff and Gay[3] presented their findings in the use of a combined denervation procedure, resecting the posterior pulmonary plexus, in the management of intractable bronchial asthma. An impressive contribution was made by Carr and Chandler[4] in 1948 in the presentation of a group of patients with intractable bronchial asthma and associated adrenalin® resistance, who showed long term benefit from the use of dorsal sympathetic ganglionectomy for intractable bronchial asthma. Also in 1948, Blades[5] presented his experiences with the application of a more radical type of resection of the pulmonary plexus in the treatment of bronchial asthma.

Although scattered interest in the direct attack upon the vagus nerve with regard to pulmonary disease had been manifested prior to 1938, the initial definitive interest in regard to the function of this nerve in relation to bronchospasm should probably be attributed to Reinhoff and Gay. They stressed a removal, as completely as possible, of the vagus nerve components in conjunction with resection of the posterior pulmonary plexus. Our group at Emory University has had a major interest in pulmonary autonomic nerve surgery since 1946, and we have reported our experiences from 1948 to date.[6–10] In 1949 Curtis and Klassen[11] made a distinct contribution to our knowledge of the physiologic activity of the vagus nerve relative to the bronchial tree in the human. In 1950 we reported further upon our experiences with vagus nerve resection and other autonomic nerve surgery in the treatment of pulmonary emphysema; and in addition we described our experiences with autonomic nerve surgery in the management of bronchial asthma, the management of recurrent spontaneous pneumothorax, in the palliation of inoperable bronchogenic carcinoma, and as an adjunctive procedure in the management of bronchospastic states associated with bronchiectasis and idiopathic bronchorrhea.

In November, 1951 we first presented our new concepts on the treatment of pulmonary emphysema and reported further upon them in 1952 and 1953. Crenshaw and Rowles[12] presented their concepts of autonomic nerve surgery in the treatment of pulmonary emphysema, including some modifications (stripping of the parietal pleura and talcum visceral pleural irritation) to the procedure which we had described.

PHYSIOLOGIC CONSIDERATIONS

A discussion relative to the surgery of the pulmonary autonomic nervous system should include a brief consideration of the causes of the confusion, and in some instances, disrepute involved in surgical procedures upon the autonomic nerve system. The autonomic nervous system in the human is a more intricate structure than the same system in the experi-

mental animal. Parallel findings in regard to the function of the autonomic nervous system do not necessarily occur in different species of animals and therefore an experiment upon a dog, or other animal, is not necessarily reproducible upon the human autonomic nervous system. It is quite possible that there may be some degree of variation of autonomic nervous system response according to the degree of development of the higher centers of the central nervous system. A final consideration must be to appreciate that the majority of the diseased states for which autonomic nervous system surgery is proposed are diseases in which there appears to be a synergism or interaction of multiple causative agencies or factors. Autonomic nerve surgery *alone* cannot be expected to alter completely diseases which are due to a combination of disturbances of autonomic nerve status and other intrinsic or extrinsic factors.

It would be well to briefly review some of the known physiologic activities of the autonomic nervous system in regard to the pulmonary bronchovascular bed. Stimulation of the vagus nerve produces both vasoconstrictor and vasodilator impulses going to the pulmonary vessels, with predominance of a dilatation effect in the dog. In the rabbit, only constriction of the pulmonary vessels results from stimulation of the vagus nerve. The autonomic nervous system is apparently responsible for a considerable number of reflexes which exist between the pulmonary and the cardiovascular systems. The vagosympathetic nerves are required to be intact to allow the reflex which produces a fall in systemic blood pressure by raising the pressure in the pulmonary circuit, in particular by raising the pressure in the pulmonary veins. The response of the heart to pulmonary ventilation has been studied by Anrep.[13] He showed that the impulses created by pulmonary inflation depressed the tone of the vagus center, with resultant increase in heart rate. This response can be destroyed only by sectioning the pulmonary vagus fibers or the cardiac vagal fibers. Slowing of the cardiac rate is produced by extreme pulmonary inflation. There appears to be an additional factor controlling these responses involved in the interaction of impulses between the respiratory and the cardioinhibitory centers within the brain. Best and Taylor[14] considered that the bronchoconstrictor fibers to the bronchiolar musculature arise from the vagus, while the bronchodilator fibers are sympathetic in origin. Efferent nerve fibers from the lungs are found in the vagus nerve. Their role in the production of sensation in the human has been well described by the work of Curtis and Klassen.[11] Brodie and Dixon[15] demonstrated that stimulation of the vagus nerve in the neck of the dog caused severe constriction of the bronchioles with resultant increase in lung volume. They also demonstrated that nerves from the stellate and adjacent thoracic sympathetic ganglia contained fibers capable of producing bronchodilatation upon stimulation. Binger, Gaarde and Markowitz[16] showed that a similar response with the intact chest could be produced by experiments upon the guinea pig. They demonstrated that stimulation of the vagus can cause such a marked closure of the bronchioles as to finally abolish the rhythmic excursions of the lung in response to insufflation. They noted a valve-like type of closure so that some air could enter, although much less could escape.

However, it is important to stress that the severe bronchospasm producible by the injection of histamine may persist in spite of bilateral transection of the vagus nerves below the level of the recurrent laryngeal nerves.[11] In this connection it is important to remember that uterine muscle fibers totally separated from nervous control, which have been previously sensitized to a specific antigen, may be caused to go into violent contraction by exposure to a specific antigen. The possibility of bronchorespiratory musculature responding directly to certain chemical agents, alterations in pCO_2 and pH should also be kept in mind.

In summary, therefore, we appreciate the over-all general tendency for the vagus nerve to produce bronchial constriction and the sympathetic nervous system to produce bronchial dilatation in the human. The exact response of the human pulmonary artery to autonomic nerve stimulation is not completely clarified; there is some evidence to suggest that vagotomy in the dog produces a lowering of pulmonary artery pressure.[17] Recent work in the human[18] has shown that injection of aminophyllin in patients with pulmonary emphysema may result in a decrease in pulmonary artery pressure. The ability of the vagus nerve to transmit the sensation of bronchial pain and bronchial irritation in the human has appar-

ently been conclusively proven. Any sensory component of the sympathetic nervous system to the lung in the human has not as yet been demonstrated.

It might be well to describe some of the experiences which we have had in work upon

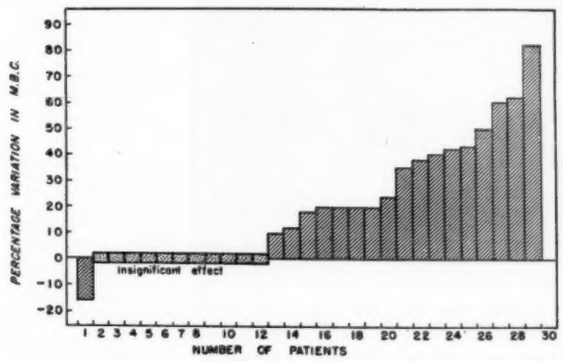

FIG. 1. Effect of aminophyllin (250 mg.) intravenously on maximum breathing capacity.

humans, which pertain directly to possible basic physiologic considerations. First, it is important to stress the difficulty of obtaining total vagus nerve denervation of the individual human lung. Our experience indicates that there is a frequent, if not constant, occurrence of branches of both vagus nerves going to the lung from a level above that of the recurrent laryngeal nerve. This is particularly impressive in regard to the left lung. It is almost constant, also, to find three or more branches coming off of the recurrent laryngeal on the left going to the left main stem bronchus. The branches above the recurrent laryngeal nerve on the left are prone to be distributed to the left pulmonary artery and superior pulmonary vein. The branches above the recurrent laryngeal nerve and from the recurrent laryngeal nerve on the right are prone to blend with the plexus of nerves about the lateral aspect of the trachea It is because of these considerations and the distinct specification by Curtis and Klassen that the denervation in their human cases was below the recurrent laryngeal nerve that we must question the validity of some of their conclusions relative to the freedom of the bronchial musculature from vagus control. It has also been our experience to note upon bronchoscopic examination a constant, apparently complete and permanent relaxation of the bronchial musculature and absence of bronchial sensitivity following complete pulmonary vagectomy on the right. It had been our policy in regard to the left lung to perform denervation by means of transection of visible fibers from the vagus nerve to the lung above the level of the recurrent laryngeal, and those from the recurrent laryngeal in conjunction with pulmonary plexectomy to achieve vagal

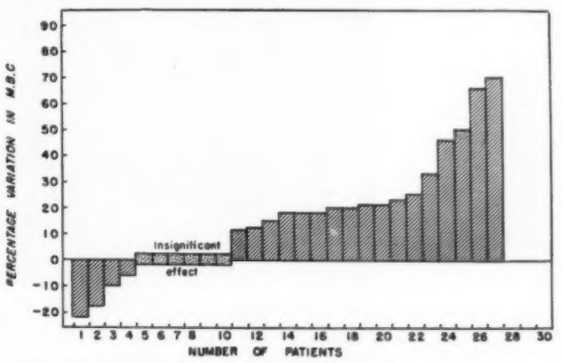

FIG. 2. Effect of adrenalin, 5 minims, by subcutaneous injection on maximum breathing capacity.

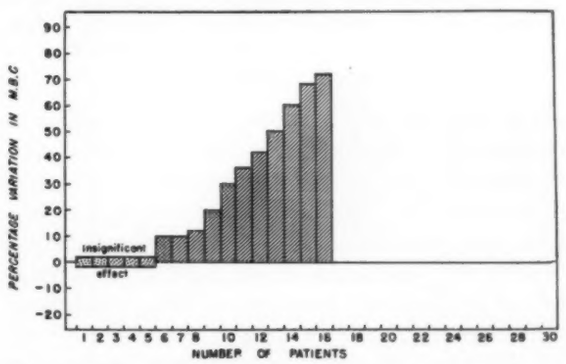

FIG. 3. Effect of ora banthine (200 mg.) on maximum breathing capacity.

denervation of the lung and yet maintain integrity of the left vagus to the gastrointestinal tract. In these patients it has been a fairly constant finding to see a relaxation of the bronchial musculature and absence of bronchial sensation which lasts from six months to one year, but we have not been able to accomplish complete and permanent denervation of the left lung by this means. It is now our policy on the left side to transect the left vagus nerve below the level of the recurrent laryngeal nerve with a complete excision of all demonstrable pulmonary fibers above that level. To date it appears that we are obtaining a more complete denervation, and probably a more permanent one; further time is necessary to substantiate this observation.

We have had to rely upon bronchoscopic visualization of a relaxed or non-spastic bron-

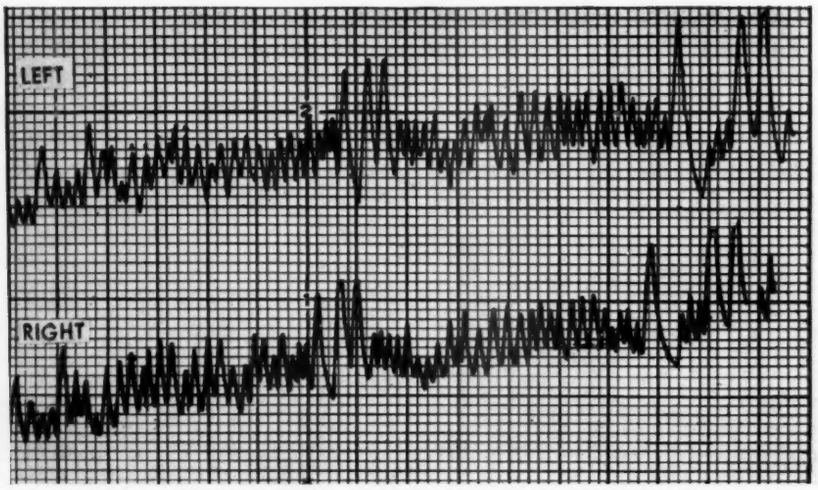

FIG. 4. Patient B. B. Bronchospirometric study carried out one year after operation on right lung. The right or previously more severely diseased lung now has the better oxygen utilization (225 cc. as against 175 cc.). Left lung: tidal air, 230 ml.; minute volume, 7,135 ml.; oxygen consumption per min., 175 ml.; carbon dioxide consumption per L. air, 42.2 ml.; vital capacity, 755 ml. Right lung: tidal air, 310 ml.; minute volume, 4,965 ml.; oxygen consumption per min., 225 ml.; carbon dioxide consumption per L. air, 45.2 ml.; vital capacity, 755 ml. About 30% of right lung resected with concomitant pulmonary vagotomy.

chial tree on the denervated side, versus a contractile, spastic bronchus on the non-denervated side, to demonstrate bronchial relaxation. By this direct method of observation one can state that there is unequivocal evidence of relaxation of the main stem bronchi and of the primary branches of the same following transection of the vagus nerve. Inasmuch as the vast majority of patients undergoing such surgery have extensive emphysema, bronchographic studies have been contraindicated. The effects of vagotomy on bronchospasm have been studied by comparative evaluation of the response of bronchospirometric tracings to the injection of various bronchodilating drugs prior to surgery (Figs. 1 to 3), and comparing the tracings to those obtained following unilateral or bilateral vagotomy. In particular, we have been using pro-banthine® for such preoperative evaluation, and more recently have been utilizing it in the intravenous form. A further method of evaluating the bronchial relaxing effect of vagotomy has been comparative cinefluorographic studies before and after vagotomy, during full inspiration and expiration activity. For several years we have noted that following unilateral vagotomy the lung which has been denervated much more rapidly extrudes air under fluoroscopic visualization so that the mediastinum shifts towards the denervated side and the diaphragm on this homolateral side achieves a much higher elevation during expiration. Such a vagotomy-produced mediastinal shift may actually be of proportions of magnitude of 2 and 3 inches in the adult. The relative opacification of the denervated lung versus the delayed opacification of the non-denervated lung, as well as the changes in position of the mediastinum and diaphragm, have been interpreted as evidence of marked decrease of bronchospasm in response to vagotomy. Measurements of pulmonary artery pressure before and after denervation have been sporadically obtained and adequate information in this regard is not yet ready for presentation. On occasions we have noted a significant decrease in pulmonary artery pressure following denervation. Figures 1 to 4 and Table 1 represent some of the human studies in these regards.

CLINICAL EXPERIENCES WITH PULMONARY DENERVATION

It is not our desire or purpose in this presentation to offer in detail our clinical experiences with autonomic denervation. We would prefer to present our present concept of the role of autonomic nerve surgery and to show some of the changing concepts occurring with clinical trial.

Bronchial Asthma. Briefly, our present opinion relative to the autonomic nerve surgery

of bronchial asthma is that it is rarely a useful procedure. We doubt that autonomic nerve surgery alone is of any practical value in the treatment of this disease. We have not personally encountered patients with true adrenalin resistance such as those presented by Carr.

localized bronchiectatic area and lobectomy was performed. Long term prevention of recurrence of bronchospastic or asthmatoid states has been noted to occur for as long as seven years. In a similar manner we have had the occasional patient with a bronchiectatic lesion

TABLE 1

STATISTICAL DATA FROM REPEATED CARDIAC CATHETERIZATION*

Statistical Data	Preoperative	Postoperative (4 months)
Systemic blood pressure	113/61 mm. Hg	95/62 mm. Hg
Pulmonary artery pressure	31/12 (20) mm. Hg	22/8 (12) mm. Hg
General blood volume	3,975 ml./m²	3,109 ml./m²
Pulmonary blood volume	874 ml./m²	598 ml./m²
Right ventricular pressure	26/6 (13.5) mm. Hg	.....
Hemoglobin	11.1 gm./100 ml.	12.2 gm./100 ml.
Hematocrit	36.7 per cent	39.4 per cent
Oxygen consumption per minute	190 cc.	270 cc.

* Demonstrating the effect of combined pulmonary denervation and excision of areas of hypertrophic emphysema.

We do believe from the long term follow-up of his group of such patients that dorsal postganglionic sympathectomy should be considered as part of the treatment of the adrenalin-resistant patient with bronchial asthma. It has been our experience and that of others that patients receiving either pulmonary plexectomy or pulmonary vagotomy have shown temporary response from one to two years of from mild to marked improvement, but persistent gain beyond the two-year period has not been manifested by any. We do believe that there is a very select group of patients with bronchial asthma, who by some are termed "pseudo-asthma." This group of patients is comprised of those we originally described as having "trigger" mechanisms. These people have bronchial asthmatic attacks which are produced by reactivation of an inflammatory component. These patients have been found to have a localized area of destructive pulmonary disease. In such cases resection of the destroyed area alone may occasionally produce cure. Our experience suggests that resectional procedures plus vagotomy are more apt to produce permanent cure of the bronchospastic state. Thus, we have a group of patients who initially had denervation procedures for bronchial asthma and in whom a persistent palliation was not obtained. Further study revealed a and bronchial asthma in whom resection alone was performed, only to be followed by recurrence of the asthmatic state. Subsequent vagotomy has then allowed long term alleviation of the bronchospastic element. If an entire lung is to be removed for such a situation, associated denervation would hardly be expected to be necessary. Mulvihill[19] recorded an instance of a patient with intractable bronchial asthma who had total destruction of one lung; the excision of such lung produced cure. We have a similar case and the bronchospirometric tracing showing the non-functional aspect of the destroyed lung acting as a trigger mechanism is represented in Figure 5. In summary, we believe that bronchial asthma is a disease with multiple causes which cannot be expected to respond to denervation procedures alone. In the adrenalin-resistant patient dorsal sympathectomy would probably be of value in conjunction with the other necessary measures to control the disease. In the patients with localized destroyed areas of the lung exhibiting a trigger mechanism potential, resection of the destroyed area plus vagotomy may be expected to produce cure. Associated denervation procedures would not be necessary in case the entire lung needed to be removed.

Inoperable Bronchogenic Carcinoma. We have nothing to add to our concept of the value

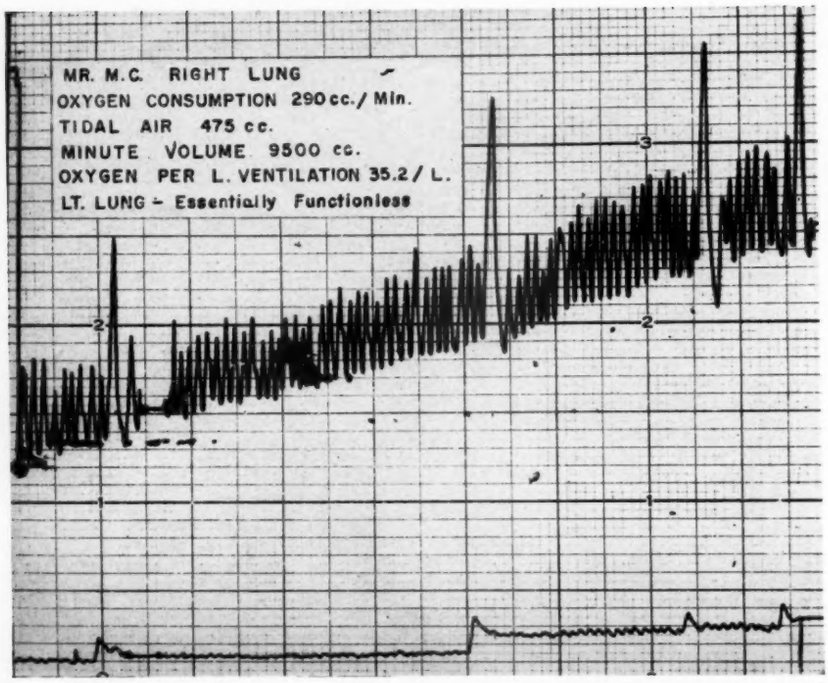

FIG. 5. Patient M. C. Bronchospirometric study demonstrating essentially entire pulmonary function on the right side. Removal of left lung provided permanent relief from previously intractable bronchial asthma. No associated denervation was performed.

of this procedure in inoperable bronchogenic carcinoma over that which we presented in 1950. Sympathectomy is of no value as a palliative procedure. Total pulmonary vagotomy, either right or left, appears to be indicated as an adjunct towards the management of intractable cough in patients with inoperable bronchogenic carcinoma who present this symptom in impressive degree. If the patient has gastrointestinal disturbances, we hesitate to recommend transection of the left vagus nerve. Transection of the right vagus nerve for inoperable carcinoma of the lung has not been associated with gastrointestinal distress in our experience.

Bronchiectasis with Severe Bronchospasm or Allergy. In this regard we believe that autonomic nerve surgery in conjunction with resectional procedures for bronchiectasis should be reserved for that group of patients who have local unilateral trigger areas combining bronchiectasis with bronchial asthma or "pseudo-bronchial asthma." In patients with bilateral bronchiectatic disease, denervation procedures would not be indicated. We would condemn any vagotomy in conjunction with unresectable areas of permanent suppurative bronchopulmonary destruction. The decrease in the efficiency of the cough reflex mechanism would be expected to allow progression of the disease in such areas. The frequency of scattered, non-discernible or non-resectable areas of bronchiectasis in patients with bilateral or far advanced disease is sufficiently high that we do not believe any autonomic nerve surgery should be associated with such types of bronchopulmonary suppuration.

Bronchorrhea. In our original communication sympathectomy was performed in two patients with bronchorrhea. Subsequent observations of these patients have shown that there has been a recurrence of the total amount of sputum production, and it is our impression that this bronchorrhea is closely correlated with major underlying emotional disturbances involved in both patients. The ultimate result in each has not been satisfactory and the use of such procedures in this condition is not recommended at this time.

Chronic Recurrent Pneumothorax. Denervation procedures as an adjunct to the treatment of chronic recurrent pneumothorax is essentially a part of the problem involved in pulmonary emphysema. It is not expected that it would be indicated in a patient with a solitary apical bleb in whom no other emphysematous

change was noted. We do believe it is indicated in patients in whom localized areas of interstitial or chronic hypertrophic emphysema are present and in whom the tendency to the bronchospastic state is demonstrable by preoperative studies. The effect of intravenous pro-banthine upon the bronchospirometric studies is evaluated prior to surgery. An increase of more than 10 per cent in the maximum breathing capacity with significant change in the oxygen consumption is at present considered indicative of some degree of hypercontractile bronchial reaction. Vagotomy, in addition to the usual reconstructive measures, is utilized in such patients. The value of such denervation procedures in this group of patients will require many years for evaluation. It is our concept that the majority of patients with hypertrophic pulmonary emphysema have the onset of the disease in a localized area of the bronchopulmonary tree. If this is true, the use of denervation procedures in the specified type of chronic recurrent pneumothorax constitutes the surgical treatment of emphysema in one of its earliest stages. This should produce a group of patients in whom future physiologic studies should be compared with such patients being treated by operative correction of recurrent pneumothorax, without the addition of vagotomy. The value of this procedure in this patient group then is at present undetermined and will await further physiologic evaluation.

Pulmonary Emphysema. We have been impressed with the frequency of pulmonary emphysema of severe debilitating degree in patients prior to the age of fifty. Emphysema as a degenerative process of advancing age has long been recognized. The frequency and importance of this presenile type of the disease deserves considerable stress. It appears to occur approximately three times more frequently in the male than in the female. Our studies suggest that 46 per cent of these patients have a significant family history relative to chronic bronchopulmonary disease and bronchospastic states. We have, further, been impressed with the incidence of prolonged bronchopulmonary irritation from single or multiple agents as a significant factor in the production of the disease. In previous communications we have emphasized the destructive potential of continued smoking or excessive smoking in individuals who respond to such a habit by cough, wheeze and so-called "chronic suppurative" or "allergic" tracheobronchitis. In a study of over 450 patients with pulmonary emphysema only 16 per cent were non-smokers, and in these an obvious other cause of continued bronchial irritation was elicited. Excessive smoking of two or more packages of cigarettes a day was noted in 33 per cent, while the remainder of the patients, namely 51 per cent, utilized about one package of cigarettes per day. In all these patients who were smoking a very dramatic change in the amount of cough and wheezing resulted whenever they were able to desist from the use of tobacco for more than three weeks. It is our conclusion that patients who smoke excessively, or in whom smoking is associated with the production of chronic cough and wheeze, are producing an intrapulmonary situation which, if allowed to continue, will produce serious degrees of pulmonary emphysema. Continuation of our studies on patients with pulmonary emphysema has shown approximately a 91 per cent incidence of increased bronchospasm to be present in this group of patients.

From a study of a large number of x-rays presented to us on patients who have been under observation for from ten to eighteen years we became impressed with the initial apparent localization of the disease in its early stages. Angiocardiographic studies made by Steinberg[20] and by ourselves on both postmortem specimens and of the pulmonary vascular bed *in vivo* have further impressed us with the apparent local destructive component of so-called diffuse, hypertrophic emphysema. Postoperative angiocardiography studies have suggested to us that a relaxation of spasm of the pulmonary arteries has been produced by combined vagotomy and dorsal sympathectomy. The possibility that such a change may be produced by vagotomy alone is now under evaluation.

In summary, it is our present concept that pulmonary emphysema of the so-called diffuse, or progressive, hypertrophic type is a disease which is resultant to an interaction or synergistic activity of several causative agents. Any of these can produce the factor of either intrinsic (inflammatory, foreign body, etc.) or extrinsic (tobacco, chemicals, etc.) irritation. This factor in sufficient amount to react with the degree of underlying or innate bronchial irritability (probably an inheritance characteristic) can produce prolonged bronchospasm and cough.

If bronchospasm and cough, which constitute continued partial and intermittently complete bronchial obstruction, are allowed to persist a sufficient length of time, alveolar overdistention with resultant decrease in bronchial arterial blood flow and an increased pulmonary artery

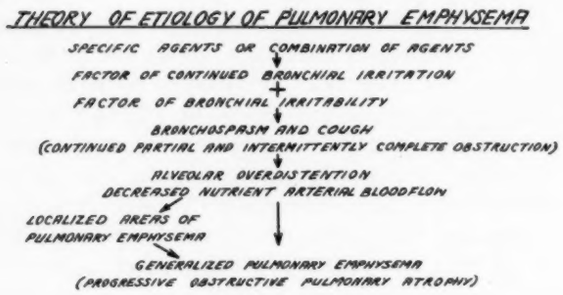
THEORY OF ETIOLOGY OF PULMONARY EMPHYSEMA
SPECIFIC AGENTS OR COMBINATION OF AGENTS
FACTOR OF CONTINUED BRONCHIAL IRRITATION
+
FACTOR OF BRONCHIAL IRRITABILITY
BRONCHOSPASM AND COUGH
(CONTINUED PARTIAL AND INTERMITTENTLY COMPLETE OBSTRUCTION)
ALVEOLAR OVERDISTENTION
DECREASED NUTRIENT ARTERIAL BLOODFLOW
LOCALIZED AREAS OF PULMONARY EMPHYSEMA
GENERALIZED PULMONARY EMPHYSEMA
(PROGRESSIVE OBSTRUCTIVE PULMONARY ATROPHY)

FIG. 6.

pressure are produced. Initial major localized areas of disease thus produced may constitute trigger areas to aid in the rapidity of the progression of the disease to produce what is generally recognized as generalized, hypertrophic pulmonary emphysema. In view of this concept we have suggested the term of "progressive, obstructive, pulmonary atrophy" as a substitute in the nomenclature for this disease. (Fig. 6.)

With such a concept of pulmonary emphysema it is apparent that attention should be paid to any surgical procedure which could constantly control or decrease the degree of bronchial irritability and bronchospasticity. Local areas of disease, if present, should be removed or corrected so that their local compressive effect and "trigger mechanism" potential is alleviated. In a recent contribution[10] we suggested a new classification for hypertrophic, pulmonary emphysema so that it might stress the etiologic factors and progressive characteristics of the disease and be capable of correlation to the type of treatment indicated according to the stage of the disease presented. Type I constitutes a chronic, diffuse, bronchospastic condition frequently typified as "chronic suppurative" tracheobronchitis, or "allergic" tracheobronchitis, in which diffuse spasm with delay in expiration is present, but no permanent destructive disease has as yet been produced. In this stage of the disease elimination of the irritant factors, either intrinsic or extrinsic, or both, should suffice. In Type II, localized, permanent, organic change in the form of hypertrophic emphysema with or without associated bullous formation is present, but localized to one area of one lung. In these patients resection of the diseased area, plus denervation procedures, has been uniformly associated with apparent clinical cure of the underlying disease. When the disease has progressed to a bilateral manifestation the problem is considerably more serious. We classify this as Type III, and subdivide it into the following groups: III*a* bilateral disease with a marked unilateral component; III*b* localized major components on both sides; and III*c* disease which appears to be completely generalized. For the most part patients with Type III*c* are beyond any real aid, either medical or surgical, except for an unusual subgroup in whom there is considerable scattered pulmonary fibrosis, and in whom there persists a major bilateral diaphragmatic activity of 2 or more inches. In this subgroup plexectomy alone has proved to be of dramatic benefit. Only six such patients have been observed, and considerable further experience will be required before a better evaluation of this aspect of the problem can be presented. We have been particularly impressed with the lack of useful response to surgical measures by the other patients in Group III*c*, the so-called generalized, diffuse pulmonary emphysema of a fixed type such that no major diaphragmatic excursion can be obtained, and no major change in maximum breathing capacity can be derived from bronchodilating drugs. In patients with bilateral disease, manifesting bilateral localized major areas of disease, the response to surgery is considerably varied and needs considerable further study. The rather select group of patients with bilateral, diffuse, hypertrophic emphysema in whom there is a major symptomatology subjectively relegated to one side of the chest, and in whom fluoroscopic observation reveals a considerable shift of the mediastinum away from the site of major disease during forced expiration, qualify for the classification of Type III*a*. Denervation procedures in this group of patients, plus excision of the major unilateral focus of disease, has produced the most dramatic response seen by us in any patients with pulmonary emphysema. Following such surgery the mediastinal shift on expiration has completely disappeared and frequently the shift is toward the side of operation, or what we termed preoperatively as

the side of "major localized intra-alveolar hypertension."

Following the studies of Liebow,[21] who demonstrated the numerous anastomoses between the bronchial arterial circulation and the pulmonary and bronchial venous bed, we believe that major doubt has been cast upon the advisability of carrying out dorsal sympathectomy procedures as part of the pulmonary denervation utilized in pulmonary emphysema. For the past year, therefore, we have treated such patients coming to surgery by vagotomy. Concomitant dorsal sympathectomy seems useful when severe pulmonary vascular sclerosis or cor pulmonale are present. The clinical response to such an approach has been quite gratifying. Physiologic studies are in progress.

FUTURE PLANS

The major factor in regard to future plans correlated to pulmonary autonomic nerve surgery is a very urgent plea for detailed physiologic objective evaluation of any such procedures in the future. It is also mandatory that one must appreciate the multiple synergistic etiologic agents involved in the majority of disease, for which such procedures are contemplated. Adequate attention to such associated factors in both the control and denervated group is mandatory. The psychic response to denervation procedures must at all times be evaluated, and not allowed to cloud scientific study o clinical results. It would appear at the present time that autonomic nerve surgery will probably come up for consideration in the management of carefully selected patients with bronchial asthma who have either proved adrenalin resistance or a local "trigger mechanism," in hypertrophic pulmonary emphysema, and as a possible method to control certain types of pulmonary hypertension, with associated cor pulmonale. In all such patients it is our present plan that they receive, whenever possible, the following preoperative evaluation: Ventilatory studies, such as the half-second expiratory curve, a three-second vital capacity and the maximum breathing capacity study should be evaluated. Bronchospirometric curves to define oxygen consumption before and after a bronchodilating drug such as pro-banthine, are obtained. The residual air must be calculated. Whenever possible, a preoperative cinefluorographic study and occasionally an angiocardiographic, or cineangiocardiographic study is obtained. Cardiac catheter studies, both prior to operation and at the time of the operative procedure, are recommended. In particular it is necessary to obtain studies of the systemic arterial oxygen saturation. The right ventricular pressure in relation to the cardiac output should be recorded. The pulmonary artery pressure should be tested at rest and in its response to regulated exercise. The irritability of the right and left bronchial tree and the degree of bronchospasticity of each must be noted at bronchoscopy. Similar studies in the postoperative period at three, six and twelve months are recommended.

SUMMARY AND CONCLUSIONS

We have tried to present the historical background to pulmonary autonomic nerve surgery. A brief summary of some of the basic physiologic concepts relative to the action of the vagus and sympathetic denervation of the bronchopulmonary structures in animals and in humans has been presented. Considerable stress has been placed upon the factor of the variation of autonomic nerve response in different species and the danger of transference of such studies to become unwarranted conclusions in regard to the function of the human autonomic nervous system. The need for further accurate physiologic knowledge of the pulmonary autonomic nervous system in the human is emphasized. A brief review of the use of pulmonary autonomic nerve surgery in the treatment of bronchial asthma, inoperable bronchogenic carcinoma, bronchospastic states associated with bronchiectasis, idiopathic bronchorrhea, as an adjunct to the treatment of recurrent spontaneous pneumothorax, and as an adjunct to the treatment of pulmonary emphysema has been presented. Multiple associated synergistic etiologic factors are involved in the production of all these diseases. Therefore, the use of autonomic nerve surgery alone without other adjunctive therapeutic measures could not be expected to produce a dramatic response or cure in any such illness. The need for objective evaluation is emphasized. A proposed plan for pre- and postoperative studies of patients undergoing pulmonary autonomic denervation is outlined. At the present time we believe we are justified in making the following conclusions:

1. The human vagus nerve appears to be the major agent for the efferent transmission of intrabronchial sensation.

2. Bronchial constriction and bronchospastic states can be produced by intermittent or prolonged stimulation of the vagus nerve.

3. Complete unilateral pulmonary vagotomy produces permanent unilateral bronchial relaxation and anesthetization.

4. Complete and permanent parasympathectomy of the left lung can only be obtained by transection of the left vagus nerve with transection of all fibers of the vagus nerve going to the lung from the recurrent laryngeal nerve and from the vagus nerve above the level of the recurrent laryngeal nerve.

5. Pulmonary autonomic nerve surgery alone is useless in treating bronchial asthma except as an adjunctive measure in those rare cases which are adrenalin-resistant.

6. Pulmonary parasympathectomy in conjunction with excision of the unilateral "trigger" area of destroyed lung can produce freedom from the asthmatic state if no extrinsic factors or allergens are concerned.

7. As an adjunctive palliative measure in the treatment of irresectable bronchogenic carcinoma, high right vagotomy may be used to control associated intractable cough. Sympathectomy has been of no value in this disease.

8. Pulmonary denervation measures are not recommended in the control of bronchospastic states associated with bronchiectasis unless such bronchiectasis is unilateral, localized, and is found to be acting as a solitary "trigger" mechanism.

9. Pulmonary denervation procedures have not proved of long term value in two cases of idiopathic bronchorrhea.

10. From a detailed study of 450 patients with pulmonary emphysema, a new and useful concept of the mechanism of the production of the disease has been derived.

11. A new classification of pulmonary emphysema has been proposed which emphasizes the etiology and the progressive pathologic physiology of the disease, and parallels the method of treatment recommended.

12. Clinical experiences with ninety-two patients undergoing autonomic nerve surgery in conjunction with other measures in the treatment of pulmonary emphysema impress us with the value of the procedure in patients in whom the disease has not reached the fixed stage. Our present concept suggests that dorsal sympathectomy is contraindicated as part of the pulmonary denervation needed for this disease. Complete high vagotomy is recommended as an adjunctive measure in all patients undergoing surgery for pulmonary emphysema of the hypertrophic type. The need for associated dorsal sympathectomy in patients with pulmonary vascular sclerosis is under study.

13. Constant adherence to certain base line pre- and postoperative studies in association with future pulmonary autonomic nerve surgery will prove useful toward: (a) more complete understanding of the basic physiology of the human pulmonary autonomic nervous system and (b) the actual usefulness and type of pulmonary denervation procedure which may prove useful for the management of pulmonary disease associated with a major component of bronchospasticity and pulmonary hypertension, or both.

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COLLINS et al. have used streptokinase-streptodornase in some thirty cases of pelvic abscess and have obtained satisfactory to excellent results. If colpotomy is performed for diagnostic and therapeutic reasons, a 1 cm. T-tube is suitable for drainage of abscess after its evacuation via colpotomy and irrigation of its sac with the streptokinase-streptodornase solution. (*Richard A. Leonardo, M.D.*)

SURGICAL REVIEW

Technic and Surgical Anatomy of Radical Neck Dissection

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IN spite of the fact that almost half a century has elapsed since radical neck dissections were introduced for the removal of cervical metastatic growths resulting from malignant lesions of the head and neck, there is nowhere in the literature known to us a detailed description of the technic and surgical anatomy of such a dissection.

The technic is at the present time considered to be relatively well standardized; nevertheless, divergent opinions still exist as to whether the spinal accessory nerve, the platysma, the omohyoid, the stylohyoid and the digastric muscles should or should not be sacrificed. Further, while the technic demands precise anatomic dissection with recognition and sparing of important nerves and vessels, the anatomy of the neck is particularly complicated. The intern, resident or surgeon desirous of refreshing his acquaintance with the anatomy of the region must often literally dig the information out of various texts, only to end up sometimes confused and discouraged. It is probably the complicated anatomic relations in the neck, which were too often viewed with trepidation by earlier surgeons, that account for the fact that surgical treatment of malignant lesions of the head and neck, especially the metastatic cervical lesions, remained for years so far behind the surgical treatment of other less accessible regions. Perhaps the late C. H. Mayo summed up the situation in 1905 when he said "a large part of abdominal work is recreation as compared with the bulk of what might be called the heavy surgery of the neck."

HISTORY

Prior to 1898 treatment of metastatic lesions of the neck remained one of the most serious unsolved surgical problems of the time. Surgical treatment was woefully inadequate, and often resulted in progressive and rapid dissemination of the malignant growth. It was not until about the turn of the century that George Crile, Sr.,[5,6] put radical neck dissections on a par with the Halsted procedure for the treatment of carcinoma of the breast. Crile was a pioneer in surgery of the neck and was quick to recognize that radical surgery is absolutely essential if the cervical metastatic growths resulting from primary malignant lesions of the head and neck are to be eliminated. Crile's report[6] on his method and results of treatment was a monumental piece of work and stands almost unique in that it remains amazingly modern even to the present time.

Crile reasoned that the close proximity of the deep cervical lymph nodes to the internal jugular vein and its branches made it imperative to sacrifice the vein by a block dissection. He proposed and practiced block excision of the regional lymphatics with sacrifice of the sternocleidomastoid, the omohyoid and part of the stylohyoid muscles, the entire internal jugular venous system, and the lymphatics and nodes in the connective tissue associated with these parts. He removed the submaxillary gland, however, only when cervical nodes were found to be involved. He also, daringly, advocated bilateral removal of the internal jugular vein when necessary and reported one case of bilateral neck dissection, contending that the vertebral and other deep veins were alone adequate to take over the venous drainage.

His contention that bilateral neck dissections were feasible, while disregarded for many years, has recently again been proved to be sound.

Crile also practiced composite block dissections with removal of a part of the mandible for lesions of the tongue and floor of the mouth. He even warned about the high incidence of hemiplegia and death resulting from ligation of the internal or common carotid arteries. His use of two nasal tubes which extended just beyond the epiglottis, with packing around the tubes, comes close to our present day intratracheal tube.

Surgical treatment of malignant lesions of the head and neck since 1906 has undergone various transitions in the form of less radical procedures (Bartlett and Callander[2]) such as suprahyoid dissection, this combined with an upper deep jugular dissection, supra-omohyoid dissection and dissection with preservation of various non-vital anatomic structures such as the spinal accessory nerve (Brown and McDowell[4]), the internal jugular vein, the sternocleidomastoid or platysma muscles, the stylohyoid and digastric muscles, and so forth. However, in recent years the pendulum has swung back once more to radical neck dissections as the surgical procedure of choice in the treatment of most cervical metastatic growths.

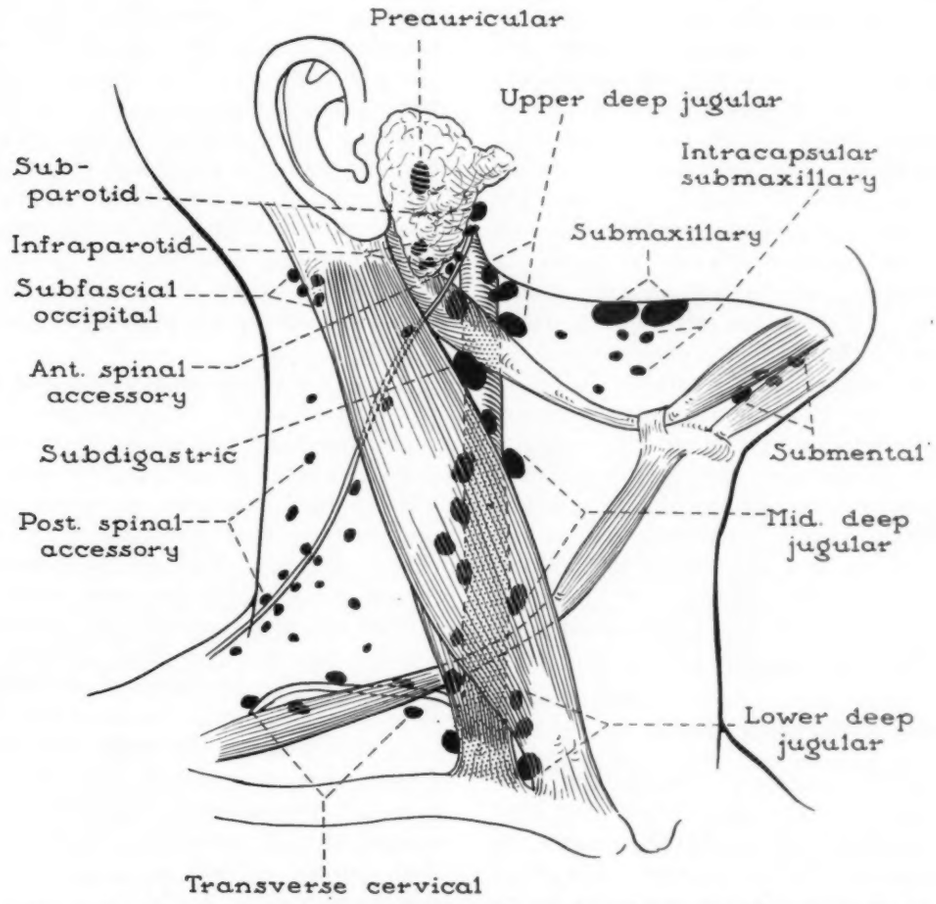

FIG. 1. A radical neck dissection for labial or intraoral carcinoma should remove all the illustrated nodes and groups of nodes except for the preauricular node.

The purpose of a radical neck dissection, in our opinion, should be the complete excision of all metastatic lesions present in the neck and the removal *en bloc* of all non-essential structures with the associated lymph nodes (Fig. 1), the lymph-bearing fascia and adipose tissue. Certain other structures remaining after a routine radical neck dissection (Fig. 6) may be sacrificed if their removal will make possible complete excision of the carcinoma.

GENERAL ANATOMY

Landmarks

Just as do most regions of the body, the neck presents certain useful anatomic landmarks; in fact it is particularly well endowed with many, and these can help the surgeon immeasurably in recognizing the somewhat complicated anatomy of the region. The bony and cartilaginous landmarks which are particularly important in radical neck dissection include the symphysis, lower border, ramus and angle of the mandible, the mastoid and styloid processes, the superior nuchal line and the external occipital protuberance of the occipital bone, the hyoid bone with its greater cornu, the thyroid cartilage, the clavicle and the manubrium sterni. The muscles particularly useful as landmarks include the sternocleidomastoid, trapezius, the anterior and posterior bellies of the digastric, the superior and inferior bellies of the omohyoid, the stylohyoid, the anterior scalene, the hyoglossus, the mylohyoid and the constrictor muscles of the pharynx. When the important nerves, arteries, veins and lymphatics of the neck are related to the foregoing landmarks, the anatomy of the neck loses some of its mystery and formidability and becomes reducible to simpler terms.

The Platysma Muscle

The so-called superficial fascia (the subcutaneous tissue) of the neck differs from that of most of the body only in that it contains the platysma muscle. This is a broad, thin sheet of voluntary muscle varying markedly in its development among individuals but typically covering the major portion of the anterolateral surface of the neck. Since it is embedded in the subcutaneous tissue, loose connective tissue intervenes between it and the deep fascia of the neck, and allows for a wide range of movement of the skin and the platysma over the deeper structures of the neck. When the platysma presents its average normal development (it may be so poorly developed as not to be recognizable), it extends from the thorax and shoulder to the mandible. It arises in the connective tissue over the upper portion of the pectoralis major and deltoid muscles, passes across the clavicle and, in the lower part of the face over the mandible, blends more or less with muscles attaching to the angle of the mouth, particularly the risorius, triangularis and quadratus labii inferioris muscles.

While the anterior borders of the two platysma muscles commonly approach each other in the midline so that they lie close together below the chin, these borders are more laterally placed at the level of the clavicle, tending to lie at about the level of the sternoclavicular joints. Because of the obliquity of the platysma and the oblique course, but in the opposite direction, of the sternocleidomastoid (which subdivides the neck into anterior and posterior triangles), the covering of the lower part of the anterior triangle of the neck does not ordinarily include platysma, while the more anterior and inferior portions of the posterior triangle are covered by platysma but the upper portion of this triangle is not (Grant[9]).

There are two schools of thought concerning the disposition of the platysma in a radical neck dissection: one school favors retention of this muscle as a part of the skin flaps; the other school routinely sacrifices the muscle, maintaining that to leave it is incomplete cancer surgery. We are proponents of the former school, believing that once superficial lymphatics are involved the carcinoma is so widely disseminated that nothing is to be gained by further surgical procedures; with superficial lymphatic involvement, the deeper vital structures of the neck, such as the common or internal carotids, are often hopelessly involved. Occasionally, however, one does see a small area of carcinoma attached to the skin that can be excised as a part of the block specimen.

Moreover, routine sacrifice of the platysma is sometimes followed by adverse effects: there is often a resultant necrosis of the skin flaps, with delayed healing and more subsequent scarring; further, the removal of this muscle necessarily results in closer attachment of the skin through scars to the underlying deep structures, including such vital ones as the carotids and the vagus nerve, and may make for uncomfortable and annoying symptoms.

Related Nerves. The platysma is innervated by the cervical branch of the facial nerve, which descends from the inferior or cervicofacial division of the facial nerve behind the angle of the mandible to reach the deep surface of the muscle and supply it. The muscle is pierced, but not supplied, by the anterior

cutaneous nerve of the neck (cutaneus colli, cervical cutaneous) and by branches of the supraclavicular nerves. The anterior cutaneous nerve of the neck emerges around the posterior border of the sternocleidomastoid and runs forward superficial to this muscle, but deep to the platysma, and either superficial or deep to the external jugular vein to divide into ascending and descending branches. The twigs from these branches pierce the platysma to reach the skin, the ascending branch usually also having an anastomosis with the cervical branch of the facial nerve. The anterior cutaneous nerve of the neck and the supraclavicular nerves (the former from C2 and C3, the latter from C3 and C4) are sacrificed in block dissections of the neck, along with all other cutaneous branches of the cervical plexus.

The supraclavicular nerves usually arise by a common trunk which then divides into three branches, named from their distribution the medial or anterior supraclavicular nerve, the intermediate or middle nerve and the lateral or posterior nerve. Their distribution includes a triangular region embracing the skin over the lower portion of the sternocleidomastoid muscle, the lower part of the posterior triangle, and skin over the tip of the shoulder and the pectoral region down to about the level of the second rib.

Deep Cervical Fascia

Deep to the platysma and the superficial fascia, and investing the neck like a sleeve, is the anterior, superficial or investing layer of the deep cervical fascia which extends from the ligamentum nuchae and spines of the cervical vertebrae completely around the neck. This fascia is attached above to the external occipital protuberance, the mastoid process and the zygoma (after surrounding the parotid gland) and, more anteriorly, to the inferior border of the mandible. Toward the anterior midline it is interrupted and divided into an upper and lower portion through the fact that it attaches to the body and greater cornu of the hyoid bone. Below, it is attached to the spine of the scapula, the acromion, the clavicle and the manubrium sterni.

Traced forward from its origin at the posterior midline, the superficial or investing layer of the deep cervical fascia passes on both sides of the trapezius muscle, crosses the posterior triangle and then splits again at the posterior border of the sternocleidomastoid to pass on both sides of this muscle. Along most of the length of the anterior border of the sternocleidomastoid muscle the two layers come together again to continue as a single layer to the midline where they are continuous with the fascia of the opposite side; however, for approximately 1 inch (about 2.5 cm.) above the level of the sternum they fail to do so. The anterior and posterior layers of this fascia between the sternal heads of the sternocleidomastoid attach below on the anterior and posterior aspects of the upper border of the sternum and enclose between them a potential space, the suprasternal space (of Burns). This space contains a small amount of fat and loose connective tissue, the lower part of the anterior jugular vein, the anterior jugular venous arch when this exists and, usually, a lymph node or two.

The superficial or investing layer of the deep cervical fascia also gives rise on its deep surface to a lamina of connective tissue which contributes to the anterolateral wall of the carotid sheath and to thin laminas which pass around the various strap muscles of the neck. The most posterior of these laminas, intervening between the thyrohyoid muscle and the thyroid gland and trachea, is continuous laterally with the carotid sheath and is the layer frequently referred to as the pretracheal fascia or the middle layer of the deep cervical fascia.

The deep or posterior layer of the deep cervical fascia arises posteriorly, like the superficial layer, from the ligamentum nuchae and the spine of the seventh cervical vertebra, and it encircles the muscles attached to the vertebral column. Thus as it is traced forward it lies at first on the outer surfaces of the splenius capitis and splenius cervicis muscles and the levator scapulae, and then on the outer surfaces of the posterior, middle and anterior scalenes, respectively. As it emerges from under cover of the trapezius, it forms the floor of the posterior triangle of the neck and is separated from the anterior or investing layer of fascia by a variable quantity of loose connective tissue and fat. That part of the fascia over the scalene muscles is frequently referred to as the scalene fascia. As it reaches the anterior tubercles of the cervical transverse processes, it becomes attached to them and then extends across the front of the vertebral column, from one set of anterior tubercles to the other, in

front of the longus muscles. Here it is frequently called the "prevertebral fascia," and a special layer of it has been referred to as the "alar fascia" (Hollinshead[10]).

Above, the deep or posterior layer of the deep fascia is attached to the base of the skull. At the base of the neck it is continued downward into the mediastinum, a portion of it between the anterior and middle scalene muscles also being evaginated as a part of the sheath of the brachial plexus.

Since lymph nodes and lymphatics are embedded in looser connective tissue lying largely between the anterior and posterior layers of the cervical fascia, and are especially associated with the fascia forming the carotid sheath, the connective tissue lodging them is removed from the anterior and posterior triangles of the neck as completely as possible during the course of a radical neck dissection for the elimination of lymph-bearing tissue. Thus the dissection is carried to or through the posterior layer of the fascia and exposes the contents of the carotid sheath. Particular pains are taken to remove the carotid sheath, and in order to do this the internal jugular vein is sacrificed.

Non-vital structures which pierce or are intimately attached to these layers (for instance, the superficial or cutaneous branches of the cervical plexus, the omohyoid muscle and even, in our technic, the spinal accessory nerve) are likewise removed in the dissection.

Triangles of the Neck

Since radical neck dissection is actually a surgical dissection of the anterior and posterior triangles of the neck for the express purpose of ablating the cervical lymph-bearing tissue *en bloc*, a short consideration of the anatomy of these individual triangles seems appropriate. Further, the somewhat complex anatomy of the neck becomes simplified when it is thought of in terms of the smaller units that the triangles present. Further details of the anatomy of these triangles, of particular interest in block dissections, are recounted in the following section dealing with the specific technic and applied anatomy.

The sternocleidomastoid muscle divides the neck into two large triangles, the anterior and the posterior. The borders of the anterior triangle are the anterior edge of the sternocleidomastoid muscle, the midline of the neck from the manubrium sterni to the symphysis of the mandible and the inferior margin of the body of the mandible. Those of the posterior triangle are the posterior border of the sternocleidomastoid muscle, the anterior border of the trapezius and the clavicle.

Anterior Triangle. The anterior triangle is subdivided into four subsidiary ones by the anterior and posterior bellies of the digastric muscle and by the superior belly of the omohyoid muscle. These four triangles are the submental, the digastric or submaxillary, the carotid or superior carotid, and the inferior carotid or muscular triangles.

Submental Triangle. The submental triangle is a small one, bounded laterally by the anterior belly of the digastric muscle, inferiorly by the hyoid bone and medially by the midline of the neck between the hyoid bone and the symphysis of the mandible. Its floor is formed by the mylohyoid muscle and it is covered by that portion of the anterior layer of the deep fascia of the neck extending between the hyoid bone and the mandible. Superficial to the deep fascia over the triangle are some superficial veins which usually contribute to the anterior jugular vein, the platysma muscle and the superficial fascia associated with that, and the skin.

Within the triangle are the terminal portions of the submental artery and vein, and a few submental lymph nodes. Although the lymph nodes are infrequently involved with metastatic cancer in its operable stages, they are more frequently involved when metastasis has occurred along superficial rather than deep lymphatic channels. For completeness of dissection, however, the contents of this triangle should be sacrificed in a radical neck dissection. This is especially true when the primary lesion is in the anterior part of the tongue, the anterior part of the floor of the mouth, the lower lip or the lower anterior portion of the gingiva.

Digastric Triangle. The digastric or submaxillary triangle is limited anteriorly by the anterior belly of the digastric muscle, posteriorly by the posterior belly of this muscle and superiorly by the lower border of the mandible. Covering it are the skin, superficial fascia, platysma muscle and deep cervical fascia; in this roof are the cervical branch of the facial nerve and a part of the ascending branch of the cutaneus colli nerve. The marginal (marginal mandibular) branch of the facial nerve may

descend below the inferior border of the mandible to run in the roof of the triangle, but often parallels the lower border of the mandible or remains entirely above it. The floor of the triangle is composed largely of the mylohyoid muscle, but behind this muscle the hyoglossus muscle contributes to a small part of the floor.

The area of loose connective tissue represented below the border of the mandible by the digastric triangle is continued both upward, medial to the jaw and backward, behind the mandibular ramus. Above the border of the mandible the extension is a part of the lateral pharyngeal space. It is bounded laterally by fascia on the deep surface of the internal pterygoid muscle, and medially by the hyoglossus and superior pharyngeal constrictor muscles. Posteriorly the extension from the submaxillary triangle is occupied by the lower pole of the parotid gland, separated from the posterior border of the submaxillary gland by the stylomandibular ligament, which extends from the styloid process to the ramus of the mandible. The potential space lying between the deep surface of the parotid gland and the lateral pharyngeal wall is also a part of the lateral pharyngeal space.

The digastric triangle is largely filled by the submaxillary gland, hence the alternative name for this triangle, submaxillary triangle. The gland also extends upward deep to the mandible, overlaps to a variable extent the two bellies of the digastric muscle and typically sends a prolongation forward, along the submaxillary duct, above the upper surface of the mylohyoid muscle. The gland is enveloped by a fascial layer formed by the splitting of the superficial layer of the deep cervical fascia, closely bound to the gland by the septa which are sent into it. Superficial to the submaxillary gland the anterior facial vein crosses the digastric triangle to reach the anterior border of the mandible, while the external maxillary (facial) artery, which at first runs upward deep to the mandible, descends into the triangle to emerge above or through the upper border of the submaxillary gland, then rounding the inferior border of the mandible and coursing upward on the face.

The submaxillary triangle is cleaned out in the process of radical neck dissection in order to remove the submaxillary lymph nodes which are located especially about the external maxillary (facial) artery and the anterior facial vein, and in front of, within and behind the submaxillary gland. This requires removal of the submaxillary gland and therefore necessitates dissection medial to the mandible above the submaxillary triangle. Dissection of the area behind the mandible is carried out also in order to expose the upper end of the internal jugular vein.

Deep to the submaxillary gland the mylohyoid nerve and the submental branch of the facial artery course forward on the external surface of the mylohyoid muscle. Behind the posterior border of the mylohyoid muscle the hypoglossal nerve has a brief course through the posterior triangle, entering this deep to the posterior belly of the digastric where it lies on the surface of the hyoglossus muscle and then disappearing deep to the mylohyoid muscle. Deep to the hypoglossal nerve in the most posterior part of the digastric triangle, or completely under cover of the posterior belly of the digastric and stylohyoid muscles, is a short segment of the lingual artery where it lies on the middle constrictor before disappearing deep to the posterior border of the hyoglossus muscle. Above the level of the lower border of the mandible but included in the area involved in a dissection of the digastric triangle, the lingual nerve runs forward on the hyoglossus muscle. It is attached to the submaxillary gland through the submaxillary ganglion, which both receives and sends fibers into the nerve and also sends fibers into the gland.

Close to the posterior border of the digastric triangle, but partly under cover of the angle and ramus of the mandible and partly deep to the parotid gland, are the external carotid artery and posterior facial vein which lie deep to the posterior belly of the digastric and the stylohyoid muscle as they run upward and are separated from the deeper lying internal carotid artery by the styloglossus and stylopharyngeus muscles.

Superior Carotid Triangle. The superior carotid triangle is bounded behind by the anterior border of the sternocleidomastoid muscle, anteriorly and inferiorly by the superior belly of the omohyoid, and superiorly by the greater cornu of the hyoid bone and the posterior belly of the digastric muscle. It has the usual coverings of skin, superficial fascia, platysma muscle and deep fascia. Passing

superficially across it are usually two large tributaries of the common facial vein, and the great auricular nerve, branches of the cervical branch of the facial nerve and branches of the cutaneus colli nerve (Lewis[12]). The floor of the triangle is formed anteriorly by the thyrohyoid muscle, posteriorly by the middle and inferior constrictors of the pharynx.

This triangle contains the upper part of the common carotid artery, the carotid bifurcation, and the first parts of the external and internal carotids. The superior thyroid, occipital and ascending pharyngeal branches of the external carotid usually arise in this triangle, and the lingual and external maxillary (facial) arteries arise either in it or just above its upper border. The arteries are crossed superficially by tributaries of the internal jugular vein, including the superior thyroid, lingual, common facial, occipital and pharyngeal veins. The nerves in this triangle include the hypoglossal nerve and its descending branch (descendens hypoglossi), the vagus and its superior laryngeal branch (divided into external and internal branches), the spinal accessory nerve, the descending cervical nerve and the ansa hypoglossi, and a part of the cervical sympathetic trunk.

Because deep cervical lymph nodes are closely associated with the fascia about the vessels in the superior carotid triangle, relationships here are of particular importance in radical neck dissection.

Inferior Carotid Triangle. The inferior carotid triangle is the least important of the cervical triangles in a radical neck dissection. Its anterior boundary is the midline of the neck, its posterior one the anterior border of the sternocleidomastoid muscle, and its superior one the superior belly of the omohyoid muscle. In its roof, consisting as usual of skin, superficial fascia, platysma and deep cervical fascia, pass branches of the supraclavicular nerves. Its floor is the sternohyoid and sternothyroid muscles. In part deep to the floor but largely overlapped below by the sternocleidomastoid muscle are the carotid sheath and its contents. Associated with the carotid sheath are branches of the ansa hypoglossi to the strap muscles, and behind it are the inferior thyroid artery, the sympathetic trunk and, medially, the recurrent nerve.

Posterior Triangle. The posterior triangle is formed by a splitting of a single embryonic muscle mass which develops into the sternocleidomastoid and trapezius muscles (Grant[9]). These muscles commonly have an attachment on a continuous line above, extending from the external occipital protuberance along the superior nuchal line to the mastoid process. The proximity of their attachments above forms the apex of the triangle, the anterior and posterior boundaries of which are these two muscles. Since the sternocleidomastoid arises from approximately the proximal third of the clavicle, while the anterior fibers of the trapezius insert on about the distal third, the base of the triangle is the middle third of the clavicle.

The immediate roof of the triangle is the superficial or anterior layer of the deep cervical fascia, with platysma muscle intervening between this and the skin only in the lower part of the triangle. The floor of the triangle is the posterior or deep layer of the deep cervical fascia as it covers the splenius, levator scapulae and scalene muscles. The posterior triangle is usually described as being divided into two subsidiary triangles by the inferior belly of the omohyoid muscle; the upper one is the occipital triangle and the lower one the subclavian.

Occipital Triangle. The occipital triangle, bounded by the sternocleidomastoid, trapezius and inferior belly of the omohyoid muscles, has emerging through it the superficial branches of the cervical plexus which appear just behind the posterior border of the sternocleidomastoid muscle, the spinal accessory nerve as it leaves the sternocleidomastoid to pass to the trapezius, and in its lower part a portion of the transverse cervical artery and vein. A portion of the occipital artery may appear in the apex of the triangle, lying on the semispinalis capitis muscle.

There are lymph nodes which are associated with the spinal accessory nerve and others, the transverse cervical chain, associated with the vessels of the same name. Deep to the fascia forming the floor of the triangle, in its lower and anterior part, is the upper portion of the brachial plexus.

Subclavian Triangle. The subclavian triangle is described as being limited above by the inferior belly of the omohyoid muscle, in front by the posterior border of the sternocleidomastoid muscle and below by the clavicle. In fact, the inferior belly of the omohyoid may parallel the clavicle in this region and a sub-

clavian triangle may have to be made artificially by pushing upward this muscle. Branches of the supraclavicular nerves cross the region of the triangle superficially. The subclavian vein, portions of the transverse cervical artery and vein, and sometimes the transverse scapular vessels (which commonly lie behind the clavicle) appear within this triangle, as do lymph nodes of the transverse cervical chain. In the floor of the triangle, covered by the deep, posterior or scalene fascia, are parts of the brachial plexus and the subclavian artery. Anterior to the triangle, under cover of the sternocleidomastoid muscle, the phrenic nerve lies on the anterior surface of the scalenus anterior, between the muscle and its covering fascia (Grant[8]).

TECHNIC OF OPERATION AND SPECIFIC SURGICAL ANATOMY

Preparation of the Patient

Blood Grouping. A determination of the blood grouping and Rh factor should always be ordered preoperatively, and at least 1,000 cc. of blood should be in ready reserve. On the average, 500 cc. of blood are needed during or immediately following radical neck dissection if the surgeon operates with care and reasonable dispatch.

Position. The patient is placed on the operating table with a pillow under his shoulders, so that his occiput rests on the edge of the upper end of the table. The head of the table is elevated to a 45-degree position.

Anesthesia. General anesthesia is preferable to any form of local anesthesia. The anesthetic agent employed may be either sodium pentothal® supplemented by curare, cocaine spray, nitrous oxide and oxygen, or gas followed by oxygen and ether. An endotracheal tube is absolutely essential with either technic. In experienced hands sodium pentothal® is the anesthetic of choice, but in less experienced ones it is probably safer to use the gaseous anesthesia. Patients who have had a previous laryngectomy are intubated by placing in the tracheal stoma a tube with a cuff to prevent aspiration of blood.

Draping of Patient. The draping of a patient for radical neck dissection is relatively simple. Two sterile towels are placed under his head; the bottom towel is left undisturbed and the top towel is draped about the head so that the ear on the side of the dissection remains completely uncovered. Two sterile towels are then placed longitudinally so as to overlap the towel about the head and extend down over the chest. Then two towels are placed across the chest with the upper border of the upper towel lying just below the clavicle.

A small sheet is folded in half and is placed transversely across the head and draped as follows: The lower border of the sheet is attached, about the middle of its length, to skin of the chin (or upper lip if the mouth is draped into the operative field) by a towel clip, and fastened to the head towel in front of the ear on the side of operation by a curved clamp. A second curved clamp is used to fasten the sheet and the longitudinal towels to the head towel behind the ear, and a towel clip is used to fasten the sheet to the underlying pillow. It is important that a sufficient amount of slack be provided to allow for rotation of the head to the opposite side. On the opposite side the sheet and the longitudinal towel are clamped to the head towel but only one clamp is required since the ear is not included in the exposed field. A towel clip is then used to fasten the sheet to the underlying pillow, as on the other side.

Following this the longitudinal towels are fastened to the cross towels on the chest on either side, where they meet at right angles. Then two sheets are placed full length so that they extend from just below the clavicle down over the patient and the Mayo stand.

THE INCISION

A T incision (Fig. 2, inset) is employed in our technic for radical neck dissections. Although other types of incisions currently in use are equally adequate, it really matters little which incision is employed so long as adequate exposure is obtained from the midline to the anterior border of the trapezius muscle and from the mandible and mastoid process to the clavicle. We remain partial to the T incision because with adequate retraction it affords us the necessary exposure with minimal postoperative scarring and consequent limitation of head movements.

The types of incision currently in use have been well depicted and described recently by Huffman and Lierle,[11] and include the following: the Y or Kocher incision, the reversed 7 incision, the T incision (Crile, Bartlett-Callander),

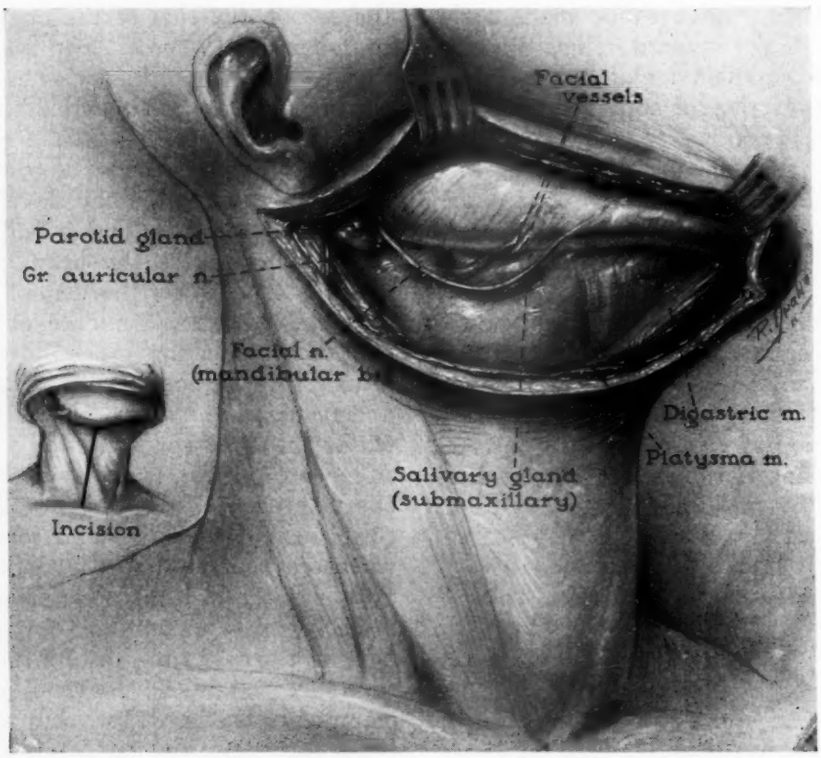

FIG. 2. A suprahyoid incision with a T extension to the clavicle (inset) is used. The suprahyoid incision is first made, and the neck structures to be removed are freed from the lower border of the mandible. The mandibular branch of the facial nerve is preserved unless the submaxillary nodes or other adjacent tissues are metastatically involved.

the T-J incision (Fischel), the Z incision (Blair-Brown), the double Y incision (Martin) and the Fraser incision.

In our technic we use plain and chromic catgut for ligature and suture material but have no objection to non-absorbable material if it is preferred by the surgeon.

Transverse or Horizontal Incision. The horizontal portion of the incision, the upper part of the T incision which we use, extends in an almost straight line from the tip of the mastoid process to the midline of the neck, paralleling and about ½ inch (approximately 1.3 cm.) below the inferior border of the mandible. (Fig. 2.) Traction is made on the skin by the surgeon and the first assistant, while the incision is carried through the skin, subcutaneous connective tissue and platysma muscle down to the enveloping layer of the deep cervical fascia. Bleeding from the submental artery and vein is encountered in the anterior portion of the incision, and the bleeding vessels are ligated. As already mentioned, we routinely include the platysma in the reflected layers and make no attempt to remove it. However, it may be sacrificed in an area overlying a metastatic mass.

Locating the Marginal Branch of the Facial Nerve. The marginal (marginal mandibular) branch of the facial nerve courses near the inferior border of the mandible and roughly parallel to it, sometimes above and sometimes below this border. It lies beneath the upper part of the platysma but superficial to both the anterior facial vein and the external maxillary artery. Since partial paralysis of the lower lip will result if this nerve is cut, it should be preserved if possible; we do this unless the submaxillary lymph nodes are involved by metastatic growths. If the nerve is being preserved, care must be taken to trace it into the parotid gland so that it is not injured as the lower portion of this gland is being removed.

After the horizontal part of the incision has been made and hemostasis has been established, the upper flap is retracted upward and elevated by two rake retractors; and a third retractor is inserted into the anterior angle of the incision in order to hold the chin over toward the opposite side. The dissection is then

carefully carried upward beneath the upper flap until the marginal branch of the facial nerve is found. The anterior facial vein is the best landmark for locating this important branch of the facial nerve, for with careful dissection the vein can be seen shining through the surrounding connective tissue, and the marginal branch of the facial nerve passes immediately superficial to it. The nerve is then traced backward to its emergence from the parotid gland and protected under two retractors. About 1 cm. behind the angle of the mandible the cervical branch of the facial nerve appears, descending to the deep surface of the platysma muscle; it may be cut with impunity.

Division of the Facial Vessels and Detachment of the Deep Fascia from the Mandible. After the marginal branch of the facial nerve has been safely removed from the field, the external maxillary (facial) artery and the anterior facial vein are clamped, divided and ligated. Since prevascular and retrovascular submaxillary nodes are closely associated with the anterior facial vein, the vessels should be divided sufficiently above the lower border of the mandible to permit removal of the nodes as a part of the surgical specimen; division of the vein at this level may involve individual ligation of tributaries which are reaching it here.

The vein lies posterior to the artery as the two structures cross the lower margin of the mandible side by side, but parts company with it immediately below the mandible, for the vein runs superficially across the submaxillary gland to join the common facial vein while the artery has a deeper course in the neck. It appears at the lower border of the mandible between this and the submaxillary gland; after its origin from the external carotid it passes upward deep to the posterior belly of the digastric and the stylohyoid muscles, and therefore also to the submaxillary gland, and then passes over or through the upper border of the submaxillary gland to reach the lateral surface of the mandible.

After the facial vessels have been ligated, their lower ends and the connective tissue associated with them are retracted downward to the lower border of the mandible; and the attachments of the deep cervical fascia to the entire lower border are detached by sharp dissection. Since this fascia encloses the submaxillary gland, detachment of it from the mandible permits removal of this gland and the other contents of the digastric triangle as part of the block dissection. (The submaxillary gland is removed both in order to insure the removal of the intracapsular submaxillary nodes which may be located within it and its fascial sheath, and to facilitate a thorough and radical block removal of all lymph-bearing tissue of the digastric triangle. Its removal is not attended by untoward sequelae.)

Removal of the Lower Pole of the Parotid Gland. The incision that separates the cervical fascia from the lower border of the mandible is carried back beyond the posterior edge of the submaxillary triangle across the lower pole of the parotid gland. This part of the gland is removed primarily to facilitate visualization of the upper end of the internal jugular vein later on in the dissection when the vein is ligated close to the base of the skull; however, its removal also eliminates the infraparotid group of nodes—one or two nodes in the tail of the parotid that usually belong to the external jugular chain. Facial paralysis will not result from cutting across the lower pole of the parotid gland at this level, for the branches of the facial nerve, except for the cervical branch, traverse the gland at a higher level; however, care must be exercised lest the marginal branch be cut here. Occasionally it loops down to this region but more commonly its lowest point is that at which it passes over the anterior facial vein. Nevertheless, the nerve should be kept in full view and retracted out of the way to prevent its accidental transection in this step. Fistulas never develop following transection of the lower pole of the parotid gland.

Related to the lower pole of the parotid gland are several structures which should be divided in order to facilitate the dissection. Before the parotid gland is transected, the external jugular vein should be identified and then clamped, divided and ligated. This vein is usually formed within or very close to the postero-inferior border of the gland by the union of the posterior branch of the posterior facial vein with the posterior auricular vein. Closely associated with the external jugular vein and roughly paralleling it, commonly just behind it but sometimes either superficial or deep to it, is the great auricular nerve which sends branches to the auricle and the skin of the mastoid and parotid regions. The nerve should be cut at this level of the dissection,

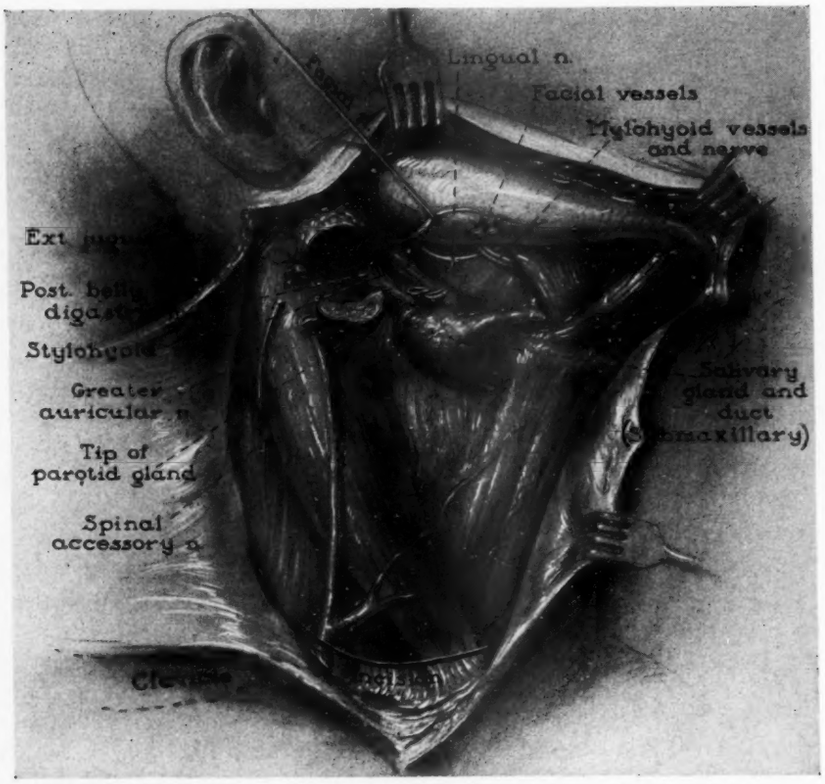

FIG. 3. After the tissues have been freed from the lower border of the mandible, the extension to the suprahyoid incision is made and the medial and lateral skin flaps are developed and retracted widely. The platysma muscle is usually preserved and left attached to the skin flaps. However, there should be no hesitancy in sacrificing this muscle or skin in places where it overlies tumor tissue.

and later on it and the stems of all the superficial branches of the cervical nerves are cut close to their origins during the removal of the block of tissue. Between the parotid gland and the submaxillary gland is a thickened portion of the deep cervical fascia, the stylomandibular ligament. This is divided as the incision through the deep cervical fascia at its attachment to the mandible is extended backward behind the level of the angle of the mandible.

After the upper or horizontal portion of the incision has been developed to this stage, it is packed with gauze and attention is turned to the vertical incision.

Vertical Incision. The vertical incision (Fig. 2, inset; Fig. 3) is made from the mid-point of the upper horizontal incision straight downward to a point on the clavicle between the sternal and clavicular heads of the sternocleidomastoid muscle. Lateral traction on the skin is made by the surgeon and the first assistant as the incision is carried down through the skin, subcutaneous tissue and the platysma muscle. Two retractors are then used to retract the anterior flap medially and elevate it. As the surgeon develops the upper portion of the anterior flap, the first assistant makes traction on the upper rake in an obliquely downward direction. Traction is an important and excellent aid to the operator in surgery of the neck, and should be used whenever and wherever possible and practicable.

As soon as the anterior flap is fully developed, to the midline and down to the clavicle, the first assistant establishes hemostasis on this flap; most of the bleeding vessels are derived from cut tributaries of the anterior jugular vein. Meanwhile the surgeon starts the development of the posterior flap, aided by posterior traction made on it with two retractors by the second assistant. As soon as the first assistant has finished clamping the bleeding vessels on the anterior flap, he shifts his attention to help develop countertraction for the posterior flap in an anterior and medial direction. This countertraction is made by

placing two sponges lengthwise on the deep cervical fascia near the angle formed by the deep surface of the skin flap and the fascia, placing all ten fingers over these sponges and pulling medially. Bleeding vessels encountered as the dissection proceeds are temporarily stopped by placing sponges over them. In this manner sponge after sponge is added, each overlapping the preceding; and as they are added, they are covered with all fingers. Through this procedure the first assistant not only provides the required tension on the flap but also helps to keep the area free of blood for proper surgical visualization. The posterior flap is developed as far down as the clavicle and carried posteriorly to the anterior edge of the trapezius muscle.

Bleeding on the posterior flap is mainly from branches of the external jugular and the posterior jugular veins. After its formation close to the lower pole of the parotid gland the external jugular vein descends approximately vertically downward on the superficial surface of the sternocleidomastoid muscle, just beneath the platysma. It reaches the posterior margin of the sternocleidomastoid muscle about 1 inch (approximately 2.5 cm.) or so above the clavicle and here pierces the deep cervical fascia to enter the posterior triangle and end by emptying into the subclavian vein. Before it ends the external jugular vein usually receives branches from the shoulder, in the form of transverse cervical and transverse scapular veins, and blood from the front of the neck through the anterior jugular vein.

The posterior jugular (posterior external jugular) vein is also a tributary of the external jugular vein, usually entering this at about its middle. It begins in the occipital region and returns blood from skin and superficial structures in the upper and back part of the neck.

The anterior jugular vein is usually formed by the confluence of small veins originating beneath the mandible, from the submental and submaxillary regions, and takes form at about the level of the hyoid bone. It communicates variably with the common facial and the internal and external jugular veins. In its fullest development it runs downward on the outer surface of the sternohyoid muscle, is united to its fellow of the opposite side across the midline by a broad anastomosis (the jugular venous arch) that lies in the suprasternal space of Burns, and then turns laterally and deeply, piercing the deep fascia and passing behind the sternocleidomastoid muscle to end in the lower end of the external jugular vein. The anterior jugular vein is, however, very variable. One or both of the two veins may be absent; they may form a vein in the midline rather than paired veins, or only the lower portions of the veins may be present, arising not from the submental region but rather by a stem of communication with the internal jugular.

Transection of the Origins of the Sternocleidomastoid Muscle

The skin flaps having been developed, the next important step is to free the origins of the sternocleidomastoid muscle (Fig. 3) from the sternum and clavicle. The sternal head of the sternocleidomastoid muscle arises from the anterior surface of the manubrium, the clavicular head from the upper border and anterior surface of approximately the medial third of the clavicle. Sometimes, however, the clavicular head is almost as narrow as the sternal head. In short, bull-necked individuals the clavicular head is likely to be markedly developed and broad, sometimes being subdivided into several slips by intervals of fascia; between clavicular and sternal heads there is usually a triangular area occupied only by fascia (Cunningham[7]).

Since the muscle has a rather extensive blood supply, including named sternocleidomastoid branches of the superior thyroid and occipital arteries (Quiring[14]), and small branches from the posterior auricular and transverse scapular arteries, transection of its lower ends is often followed by the appearance of annoying bleeding. Therefore, in our dissection the two heads of the muscle are transected in the following manner: The fascia enveloping the muscle is incised just anterior and posterior to the borders of the muscle, about 1 inch (approximately 2.5 cm.) above the clavicle. The surgeon inserts his right index finger into the anterior incision, passing his finger behind the muscle between it and the posterior lamella of the fascia surrounding the muscle. The muscle is thus freed on its deep aspect from the enveloping fascia and from important deep structures which lie just behind it.

Absolute assurance must be obtained that none of the structures of the carotid sheath (internal jugular vein and common carotid artery, vagus nerve), nor the phrenic nerve,

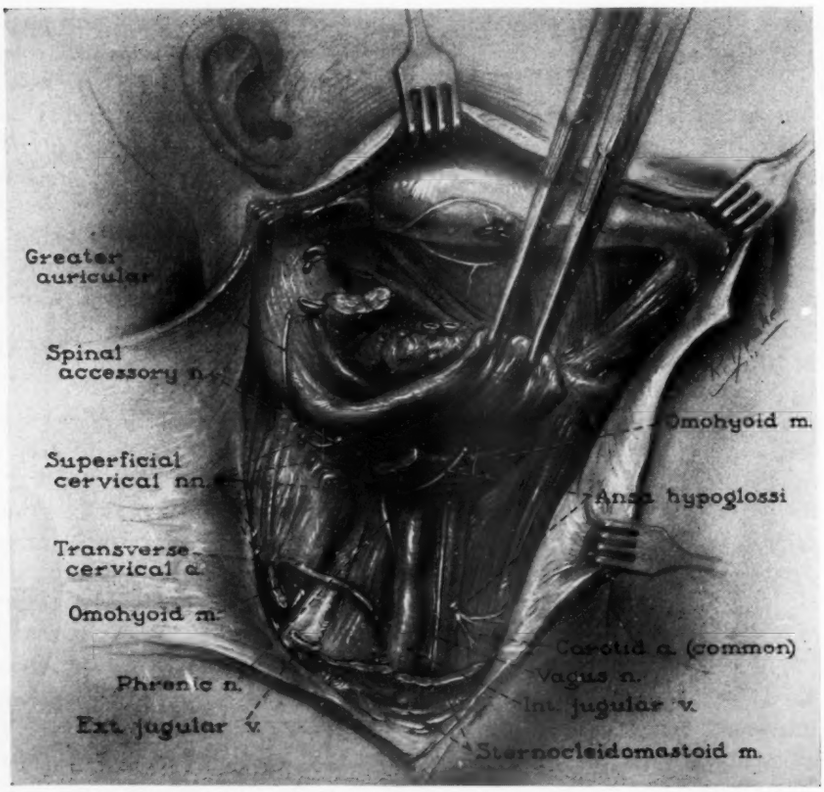

FIG. 4. The sternocleidomastoid and omohyoid muscles have been divided, and the contents of the posterior cervical triangle have been retracted medially and upward, exposing the brachial plexus, the scalene muscles and the large vessels of the neck. Usually the transverse cervical artery is sacrificed with the specimen.

have been loosened from their posterior attachments and brought forward with the muscle. If inspection of the posterior surface of the muscle does not show muscle fibers clear of fascia, the structures just mentioned should be visualized in order to be sure that they are not attached to the posterior surface of the muscle. This assurance having been attained, a large curved clamp is applied to the lower end of the sternal head as close to the manubrium sterni as possible, with the convexity downward, and another clamp is applied to the muscle about 1 inch (approximately 2.5 cm.) or so above the manubrium sterni. The muscle is transected above the lower clamp at a distance sufficient to leave a small cuff of tissue about which a ligature may be applied without danger of the muscle remnant slipping from the ligature. The clavicular head is treated similarly, and the muscle is put on a stretch and retracted upward. (Fig. 4.) Because of its size, however, the sternal head can usually be tied off in a single ligature; however, the broader clavicular head may require two or more ligatures.

Dissection of the Posterior Triangle

The next part of the dissection concerns the structures of the posterior triangle of the neck. (Fig. 4.) The spinal accessory nerve runs obliquely across it, between the superficial or investing and the deep or posterior layers of the deep cervical fascia, and is an important landmark in dissections of this triangle. Above the nerve the triangle can be dissected to its muscular floor without any particular danger, but below the nerve a number of important structures lie on the muscular floor of the triangle just beneath the deep layer of fascia covering this floor. These structures include the anterior rami of the various upper cervical nerves shortly after they leave the intervertebral foramens, and therefore motor nerves to the levator scapulae and to the scalenes (typically buried in the muscles); the roots of the brachial plexus as they emerge between

anterior and middle scalenes, and the nerve to the serratus anterior after it reaches the superficial surface of the middle scalene; the phrenic nerve, on the anterior surface of the anterior scalene; and the subclavian artery and vein. This region below the spinal accessory nerve has been called the "danger zone" of radical neck dissections, and rightly so. Dissection carried deep to the posterior layer of the deep fascia necessarily uncovers these structures, and any sharp dissection here is therefore fraught with danger.

Since it is the areolar tissue in the triangle between the anterior and posterior layers of deep fascia that contains most of the lymphatic tissue, it is particularly important to remove this as a part of the block; in this tissue and just deep to it lie arteries, veins and nerves, some of which are sacrificed and some of which must be spared. Therefore the technic of dissection of the posterior triangle is largely one of blunt dissection with the finger, supplemented by scissors when necessary, along the cleavage plane which lies between the tissue of the triangle and the underlying deep layer of cervical fascia. This blunt dissection begins along the posterior border of the sternocleidomastoid muscle and is first used to uncover the external jugular vein. The vein is followed into the depths of the posterior triangle and clamped as close as possible to the subclavian vein. The latter vein is easily "tented up" by a clamp; therefore in ligating the external jugular it is important to take pains not to incorporate the subclavian vein also. Usually the transverse scapular, transverse cervical and anterior jugular veins enter the external jugular near its union with the subclavian.

The omohyoid muscle is next uncovered, as it emerges from beneath the posterior edge of the sternocleidomastoid muscle. The connective tissue sling which holds the inferior belly of the omohyoid muscle to the clavicle is freed by blunt dissection from its lower attachment so that it and the muscle can be pushed upward, and the muscle is transected as low as possible. In so doing, two clamps are applied, one as far laterally and low as the muscle can be exposed, and the other about ½ inch (approximately 1.3 cm.) above it. The muscle is cut just above the lower clamp and ligated, and the upper end of the muscle is retracted upward as part of the block specimen. The transverse cervical and transverse scapular veins, which typically cross anterior to the inferior belly of the omohyoid to enter the external jugular vein, are clamped, cut and ligated so that their proximal ends can be included in the block specimen with the external jugular.

In order to free the base of the block in the posterior triangle the blunt dissection is carried anteriorly into the area which once lay deep to the sternocleidomastoid, to the level of the carotid sheath. It is in this maneuver that the most important structures at the base of the neck are encountered. Although they all lie behind the posterior layer of deep fascia and are therefore not disturbed by blunt dissection which separates areolar tissue from this fascia, they are more or less plainly perceived during the course of the blunt dissection and their position and relations should be appreciated. A description of them is therefore included herein.

Structures in the "Danger Zone." Nerves to the Rhomboids and the Serratus. Through or posterior to the middle scalene muscle in the posterior triangle and below the level of the accessory nerve, the nerve to the rhomboids (from C_5 close to the intervertebral foramen) may appear and run a backward and downward course, or it may be entirely hidden deep to the posterior scalene and levator scapulae. The nerve to the serratus anterior regularly attains a position just deep to the deep cervical fascia, on the anterior surface of the middle scalene, by piercing this muscle; the root so doing is derived from the fifth and sixth cervical nerves, primarily the latter, and is frequently joined by a contribution from the seventh cervical nerve which runs around the posterior border of the middle scalene. The nerve then runs downward on the scalene between this and the deep layer of the cervical fascia to attain the outer or superficial surface of the anterior serratus, the uppermost slip of origin of which, from the first rib, forms a posterior and inferior part of the floor of the posterior triangle.

Brachial Plexus. Between the middle and anterior scalene muscles the brachial plexus emerges, also lying behind the deep layer of fascia (often called "scalene fascia" here), and carrying down with it into the axilla a continuation of this fascia which helps to form the axillary sheath. The parts of the plexus appearing in the posterior triangle are the three trunks, upper, middle and lower, of which the

upper one lies most superficially, and the anterior and posterior divisions. The roots of the plexus, consisting of the anterior rami of the fifth cervical through the first thoracic nerves, are largely buried between the anterior and middle scalenes, while the cords are formed as a rule in the upper part of the axilla or at least behind the clavicle.

Subclavian Vessels and Their Branches. Associated with the lower part of the brachial plexus in the neck is the third part of the subclavian artery. This artery is separated from the corresponding vein by the anterior scalene muscle, its second part being the portion behind the anterior scalene and its third that between the lateral border of the anterior scalene and the upper border of the first rib (Brash[3]). Although the third part of the subclavian artery is usually described as typically giving off no branches, either the transverse scapular or the transverse cervical arteries may arise from this part instead of from the thyrocervical trunk (and therefore indirectly from the first part). This origin of the transverse scapular has been said to occur in from 22 to 28 per cent of cases (Hollinshead[10]). Origin of the whole or some branch of the transverse cervical artery (a part of this artery sometimes being termed the superficial cervical) has been reported to occur in as high as 30 per cent of cases.

If either of these arteries has the more lateral origin, from the third part of the subclavian, it may run in front of, through or behind the brachial plexus; if they arise from their more common origin, the thyrocervical trunk, they pierce the posterior layer of deep cervical fascia to pass in front of the anterior scalene muscle (and the phrenic nerve on this muscle) and in front of the brachial plexus. In the posterior triangle the transverse cervical artery typically runs above the level of the upper border of the clavicle, while the transverse scapular artery runs behind the clavicle; both arteries disappear laterally deep to the trapezius muscle or the clavicle.

Phrenic Nerve. Still farther forward, again behind the posterior layer of the deep cervical fascia and directly on the muscle fibers of the anterior scalene muscle, is the phrenic nerve. In this position it lies more or less under cover of the lateral border of the carotid sheath. The nerve usually arises from anterior rami of the third, fourth and fifth cervical nerves, the three contributions typically rounding the lateral border of the muscle and joining in a variable fashion on its anterior surface. A particularly low junction of one of these contributions, or sometimes even one from the sixth cervical nerve, with the main trunk of the nerve is usually referred to as an accessory phrenic nerve since it may parallel the main phrenic nerve for some distance, to unite with it only at the base of the neck behind the subclavian vein or in the superior part of the mediastinum. Sometimes an accessory branch of the phrenic nerve pierces the fascia to loop in front of the subclavian vein before joining the rest of the nerve, and occasionally, but rarely it is said, the entire phrenic nerve may pass anterior to the subclavian vein (Grant[8]).

The phrenic nerve is usually preserved in radical neck dissections; however, there should be no hesitancy in sacrificing it if such a sacrifice is considered necessary to circumvent the malignant growth. This necessarily causes partial paralysis of the corresponding side of the diaphragm.

Freeing the Posterior Border of the Block. The technic which we use for freeing the tissue of the posterior triangle along the posterior border of the triangle, so that it may be included in the block dissection, is intended to remove as completely as possible the lymphatic chains which extend laterally beneath the trapezius muscle. There are two of these; (1) the transverse cervical chain, associated with the vessels of the same name, and (2) a posterior part of the spinal accessory chain, associated with the spinal accessory nerve especially in its course between the anterior border of the sternocleidomastoid muscle and the point where this nerve disappears beneath the anterior border of the trapezius.

Lymphatic Tissue of the Posterior Triangle. Of the two lymphatic chains in the posterior triangle, the transverse cervical one is probably the least important. It consists of from one to ten nodes which usually lie between the transverse cervical vein and artery, therefore deep to the vein and superficial to the artery, and like the vessels are arranged horizontally. Its drainage joins the lower nodes of the internal jugular chain beneath the sternocleidomastoid muscle. The transverse cervical chain receives some of the drainage from the spinal accessory chain, some from subclavian nodes and some from skin of the lower anterolateral region of

the neck, and the anterior thoracic wall and superior extremity. The supraclavicular nodes of this group in the posterior triangle are more frequently involved by metastatic carcinoma from structures below the clavicle, such as the breast, lungs, kidneys, stomach, lower part of the gastrointestinal tract, testicle and even ovary, than from the neck itself. This is true also of some lower parts of the spinal accessory chain, but in order to insure complete removal of the deep cervical lymphatics of both anterior and posterior triangles these two chains should be sacrificed in continuity with the more important internal jugular chain.

The spinal accessory chain of lymph nodes consists of some five to ten which are intimately associated with the nerve, especially after it reaches the sternocleidomastoid muscle and as it crosses the posterior triangle of the neck. They receive connections from the occipital, mastoid and suprascapular nodes, from the parietal and occipital regions of the scalp, from the nape of the neck and from the lateral region of the neck and shoulder (Rouvière[15]). The uppermost nodes of the spinal accessory chain become associated with the highest nodes of the internal jugular chain; the lowest nodes, deep to the trapezius, mark the junction of the spinal accessory chain with the transverse cervical chain.

Although we recognize that the nodes of the spinal accessory group, like the supraclavicular nodes of the transverse cervical group, are much less often involved by metastasis from carcinoma of the head and neck than are the nodes of the internal jugular group, we believe that for complete and radical cancer surgery these nodes must be removed. In order to do this completely it is necessary to sacrifice the spinal accessory nerve. A particularly important consideration is the fact that before the accessory nerve enters the posterior triangle it passes through the area of loose areolar tissue which contains the very important subdigastric node of the internal jugular chain (Martin and associates[13]). This node is important because of its frequent metastatic involvement in oral cancers. Because of this, especially, we believe that some disability (in the form of drooping of the shoulder or poor abduction of the arm above the horizontal level) is a relatively small price to pay compared with the risk of recurrence of carcinoma, and that the routine practice of preserving this nerve should be inexorably condemned as incomplete cancer surgery. In our own dissections, therefore, we routinely transect the spinal accessory nerve as it passes beneath the anterior edge of the trapezius muscle. At a later stage in the dissection we transect it again in the anterior triangle so as to remove this nerve and the lymph nodes associated with it as a part of the block dissection

Aside from transverse cervical and accessory chains of nodes, there are usually none other in the posterior triangle of the neck except for a single one (subfascial occipital node) which lies high up in the apex of the posterior triangle, on the splenius capitis and beneath the fascial roof of the posterior triangle.

Technic of Freeing Tissue at the Posterior Border. After the base of the posterior triangle has been cleaned out, dissection is started along the posterior border of the triangle, working from the clavicle upward. If this tissue is simply put on a stretch and divided by sharp dissection, its posterior edges retract under the trapezius muscle and make hemostasis more difficult to obtain. The method described herein avoids this and also provides, we believe, clean and thorough eradication of lymph-bearing tissue along the posterior border of the triangle and under cover of the anterior part of the trapezius.

By means of the surgeon's left index finger the tissue bordering the posterior edge of the triangle is put on a stretch. Maintaining this tension, the tissues along the anterior border of the trapezius are systematically buttonholed by scissors, clamped, divided and ligated. The surgeon applies his clamp as close to the anterior border of the trapezius as possible while the first assistant applies a second clamp about ¼ inch (approximately 0.6 cm.) anterior to the first clamp. Then the tissue between the clamps is cut close to the posterior clamp. The posterior portion is ligated, and the anterior clamp is retracted forward and upward as part of the block of tissue to be removed. This is repeated along the anterior border of the trapezius muscle, working upward.

As the dissection proceeds, the spinal accessory nerve is encountered as it passes deep to the anterior border of the trapezius muscle; it has at this level already supplied the sternocleidomastoid and therefore consists here of fibers to the trapezius muscle alone. Running in the accessory nerve, or paralleling it as separate

branches, are nerve fibers from the cervical plexus (C_2, usually, and C_3 and C_4) which constitute the afferent innervation of the trapezius muscle. For reasons already mentioned we routinely sacrifice the spinal accessory nerve, cutting it during the dissection of the posterior triangle as it passes deep to the trapezius muscle. If there is any doubt concerning the identity of the nerve, gentle pinching with the tissue forceps will produce a movement of the shoulder, from contraction of the trapezius, if the nerve so pinched is the spinal accessory.

Above the level of the accessory nerve no structures of importance are encountered along the posterior border of the triangle until close to the apex; here the occipital artery may appear as it turns upward onto the scalp. In the apex of the triangle, also, is a subfascial occipital node.

When the tissue in the posterior triangle is entirely freed along the posterior border of the triangle, according to the method described heretofore, there will often be a large accumulation of clamps on the surgical specimen. The tissue contained in the clamps should be ligated and the clamps removed; often the tissue between from two to four clamps can be included in one ligature. This frees the specimen of unnecessary clamps so that the tissue can be retracted upward more effectively. (Fig. 4.)

Dissection Deep to the Sternocleidomastoid

Transection of the Lower End of the Internal Jugular Vein. The carotid sheath surrounds the internal jugular vein, the common carotid artery and the vagus nerve. The anterior lamina of the sheath is derived from that part of the superficial or anterior layer of deep fascia lying deep to the sternocleidomastoid muscle. The posterior lamina is derived from the deep or posterior layer of the deep fascia. The medial lamina, separating the contents of the sheath from the trachea and esophagus, is formed by a septum uniting these two laminas. The carotid sheath is well developed between the levels of the subclavian vein and the carotid bifurcation; below and above these levels, respectively, it is not so clear-cut, and opinions differ as to whether the sheath should be described as extending into the mediastinum below or to the skull above. Within the sheath the vein lies laterally and may be also somewhat anterior to the common carotid artery; behind both the vein and the artery, somewhat in the interval between them, is the vagus nerve. The three structures occupying the sheath are separated from each other by thin laminas of connective tissue.

In order to expose the internal jugular vein, the carotid sheath is opened by splitting its anterior wall transversely. The internal jugular vein is then isolated at the level of the clavicle preparatory to ligation. Two large curved clamps are applied as low down at this level on the clavicle as possible, with the convexity of the clamps pointing inferiorly; a third clamp is applied above this level. The vein is transected just above the middle clamp and its lower end is doubly ligated; the upper end is also ligated, the clamp being left on this part so that the vein can be put on upward traction with the rest of the specimen. (Fig. 5.) The vein can then be separated from the other carotid sheath structures by blunt finger dissection.

Thoracic and Right Lymphatic Ducts. In the area just posterolateral to the vein there is a mass of areolar tissue which contains the rather friable thoracic duct on the left side, or the right lymphatic duct if it is the right side. This area should be dissected with care in order to prevent injury to these ducts. They are thin-walled and often are the color of the areolar tissue surrounding them. The terminal portion of the thoracic duct may be distended with blood since the bicuspid valve with which this part of the duct is provided may be located not at the ostium of the duct into the venous system, but some ½ inch (about 1.3 cm.) from this ostium (Martin and associates[13]).

On the left side, internal jugular, subclavian and bronchomediastinal lymphatic trunks typically open into the thoracic duct, but any one of them may open independently into the venous system; on the right side these three ducts may unite to form a right lymphatic duct, but commonly one or more of them opens independently into the venous system. The thoracic duct on the left side (occasionally this duct opens into the veins on the right side of the neck instead of the left) and the right lymphatic duct or its component parts on the right side typically empty into the junction of the internal jugular and subclavian veins in the lateral angle formed by these veins. However, they or parts of them may empty instead into the subclavian vein

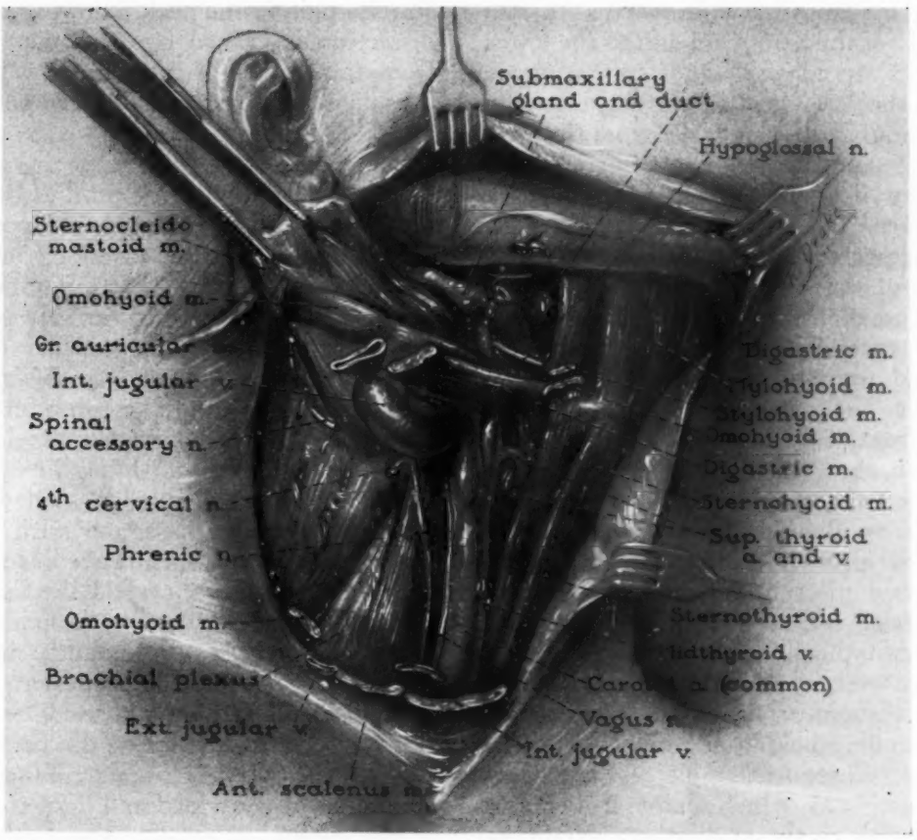

FIG. 5. The internal jugular vein has been divided at the level of the clavicle and elevated upward. The digastric muscle and contiguous structures have been freed from the mandible and retracted laterally. In the dissection the mylohyoid muscle is retracted medially to permit division of the submaxillary duct.

or into the internal jugular vein directly. Thus it may happen that some lymphatic channel empties into the internal jugular vein above the ligature, and that in retracting the tissue upward this vessel is torn. If this fact is recognized, the trunk should be ligated in order to prevent leakage of lymph.

It should also be remembered that the thoracic duct may loop relatively high in the neck before ending in the venous system. It passes behind the carotid sheath and in front of the anterior scalene muscle and its fascia to reach the lateral angle of the jugular junction, turning down in front of the upper border of the subclavian artery in order to do so. It is in this part of its course that it may show considerable redundancy, extending sometimes even 4 cm. above the clavicle (Anson and Maddock[1]). It is generally recognized that if the thoracic duct is injured in the neck, it should be ligated to prevent the leakage of lymph. Apparently, other channels—probably the right lymphatic duct—readily take over the lymphatic drainage from the thoracic duct when this is done.

Ansa Hypoglossi. As the internal jugular vein is stripped up by blunt dissection, the ansa hypoglossi usually comes into view. This nerve loop typically lies on or is embedded in the carotid sheath, but varies in position. Sometimes its two limbs, the descendens hypoglossi and the descendens cervicalis, descend medial and lateral to the internal jugular vein and therefore make a loop about it, while sometimes the descendens cervicalis runs, like the descendens hypoglossi, between the internal jugular vein and the common carotid artery. Both contributions to the ansa hypoglossi actually consist of cervical and not hypoglossal nerve fibers, and the loop innervates most of the strap muscles of the neck. A nerve to the upper belly of the omohyoid and to the upper parts of the sternohyoid and sternothyroid muscles usually arises from the

descendens hypoglossi, while nerves to the inferior belly of the omohyoid and to the lower part of the sternohyoid and sternothyroid arise from the loop itself. The nerve to the thyrohyoid muscle does not arise from the ansa hypoglossi but rather from the hypoglossal nerve shortly after the descendens hypoglossi has been given off; like the descendens, it consists of cervical nerve fibers.

Branches of the Thyrocervical Artery. As the lower end of the internal jugular vein is lifted and carried upward, the transverse scapular and transverse cervical arteries, the latter above the former, are usually seen crossing in front of the anterior scalene muscle. One or both, however, may arise from the third part of the subclavian, lateral to the anterior scalene. Behind these arteries and the fascia on the anterior scalene is the phrenic nerve. Directly adjacent to the medial border of the anterior scalene is the thyrocervical trunk, which typically gives rise to the two arteries just mentioned; from it also arises the inferior thyroid artery, which can usually be seen ascending behind the carotid sheath. This artery subsequently pierces the prevertebral fascia, at which point it bears a variable relation to the sympathetic chain. It then turns downward to enter the thyroid gland, having a variable relation to the recurrent nerve where artery and nerve cross each other.

Superficial Branches of the Cervical Plexus. Lateral to the internal jugular vein and along the posterior border of the sternocleidomastoid muscle, the tissue of the block dissection can be stripped readily from deeper structures as far up as the middle third of the sternocleidomastoid. Here it is bound down by the superficial branches of the cervical plexus, most of which emerge through the deep or posterior layer of deep fascia to pass around the posterior border of the sternocleidomastoid muscle. The lowest nerves encountered here are the supraclaviculars which, since they are passing downward across the posterior triangle, are already included in the block. Above these are the emerging stems of the anterior cutaneous nerve, the great auricular and the lesser occipital. As they are encountered, these nerves are sectioned in order to free the block. This accounts for the numbness in the region of the ear and neck that follows radical neck dissection and which in the main is permanent. Rarely, pain in the neck following radical neck dissection is caused by neuromas developing from transection of these sensory nerves, and occasionally a patient will complain bitterly of pain in this region.

Dissection in the Anterior Triangle

In the anterior part of the dissection as it is carried upward in the anterior triangle of the neck, it will be found that a number of tributaries of the internal jugular vein enter it and bind down the vein; those which do so must be divided and ligated. As the specimen is freed here, also, the carotid bifurcation and some of the branches of the external carotid artery come into view. (Fig. 5.)

As the internal jugular vein is stripped upward by blunt dissection with the finger, the first tributary encountered, and the only one until about the level of the upper border of the thyroid cartilage, is the middle thyroid vein. This vein, not constantly present and varying in size when it is present, leaves the thyroid gland at about its middle and passes transversely, superficial to the carotid artery, to join the internal jugular. When it is encountered, it is divided and ligated.

The tissue originally about the internal jugular vein and that lateral to it which extended deep to the sternocleidomastoid muscle is likewise stripped upward by blunt finger dissection, this dissection being carried laterally and posteriorly into continuity with the tissue already freed posteriorly. The superior belly of the omohyoid muscle, which is included in the block, is exposed by slitting the fascia along the anteromedial edge of the muscle, and the upper part of the muscle is stripped to its attachment on the hyoid bone, from which it is then freed.

Higher Tributaries of the Internal Jugular. The tributaries of the internal jugular vein in the region between the superior border of the thyroid cartilage and the lower border of the posterior belly of the digastric muscle vary but always include the common facial; the other veins, the superior thyroid, lingual and pharyngeal and laryngeal, may empty independently into the internal jugular or may join in various combinations, of which the most common is the drainage of all of them into the common facial vein. Since the deeper ones are sacrificed, regardless of their identity, their accurate identification is not necessary

in a block dissection and, indeed, because of the variations encountered, can usually not be done. However, a brief description of them is included herein.

The first of the several tributaries of the internal jugular vein usually encountered at about the upper border of the thyroid cartilage is the superior thyroid vein. It enters the anteromedial surface of the internal jugular and usually receives laryngeal veins. The vein is ligated as close to its source as possible, while the superior thyroid artery adjacent to the vein is left intact if feasible. The external branch of the superior laryngeal nerve (to the cricothyroid muscle) lies very close to the artery and should be avoided in any case. Except at their upper ends both of these vessels are largely under cover of the strap muscles of the neck in their course to the thyroid gland. Frequently the superior thyroid vein does not enter the internal jugular directly, but rather enters the lower end of the common facial vein. However, the stem of the facial vein between the superior thyroid vein and its termination in the internal jugular is often too short for ligation, so that usually the superior thyroid and common facial veins must be ligated separately.

The common facial vein is formed by the confluence of the anterior facial vein and the anterior branch of the posterior facial vein below the angle of the mandible, superficial to the submaxillary gland or just posteroinferior to it. Of these two components, the anterior facial vein, which was in the early part of the dissection ligated and divided on the mandible, drains the major part of the face. Its upper end, the angular vein, communicates with the cavernous sinus through anastomoses which it has with the ophthalmic vein, and with the pterygoid plexus of veins through the deep facial or buccinator vein accompanying the buccinator artery. Below the level of the mandible this vein receives branches from the submental region, or communicates with the anterior jugular vein here, and then runs downward and backward superficial to the submaxillary gland, thus diverging from the external maxillary or facial artery which it accompanies on the face.

The posterior facial vein is formed by the union of branches corresponding to those of the superficial temporal artery, joined by the internal maxillary vein or veins. It descends at first deep to, then through the parotid gland; and as it emerges at the lower pole of the gland, it divides into two stems. Of these, the posterior stem forms a large part of the origin of the external jugular vein, while the anterior stem runs downward and forward to join the anterior facial vein. Like the anterior facial vein, the posterior facial lies as a rule decidedly superficial to the arteries to which its branches correspond, for it usually runs superficial to the stylohyoid and digastric muscles, although it may lie deep to them, while the upper part of the external carotid regularly lies deep to the muscles.

Before joining the internal jugular vein, the common facial vein usually sends a communication to the external jugular, and this is sometimes the larger part of the vein. Since the common facial vein lies superficially and is included in the block of tissue to be removed, it does not have to be ligated and sectioned; rather it is the deeper branches that come into it or the internal jugular which must be so treated. In addition to the superior thyroid vein, these tributaries are usually one or more pharyngeal veins which drain the pharyngeal plexus laterally at this point (the plexus also drains up toward the pterygoid plexus and down toward the thyroid veins); one or more veins from the tongue, including the vena comitans of the hypoglossal nerve and more deeply situated veins, which may or may not unite to form a single lingual vein (in either case the veins from the tongue may empty into either the common facial or the internal jugular directly); and sometimes the occipital vein, although this more commonly ends in the suboccipital region.

Internal Jugular Lymph Nodes. The block of tissue freed up to this stage of the dissection contains not only the nodes of the posterior triangle (the transverse cervical chain, the spinal accessory group of nodes and the subfascial occipital node) but also the deep cervical nodes associated with the internal jugular vein. The latter nodes, collectively known as the "internal jugular lymphatic chain," are located chiefly along the anterior and lateral aspects of the vein and extend from close to the jugular foramen to the junction of the internal jugular and subclavian veins.

The nodes of the internal jugular chain below the level of the posterior belly of the digastric are sometimes divided simply into upper and

lower groups (Schaeffer[16]), with the omohyoid muscle constituting the line of division between them, or sometimes (Rouvière[15]) are divided into three groups, i.e., superior, middle and inferior. According to the latter division, the superior includes nodes between the lower border of the posterior belly of the digastric muscle and the thyrolinguofacial trunk; the middle group, nodes between the level of this trunk and the superior belly of the omohyoid muscle; and the inferior group, those between this muscle and the termination of the internal jugular vein. The nodes of the internal jugular chain commonly vary in number (from approximately fifteen to thirty) and in size (the more numerous they are, the smaller they tend to be).

The more constant, usually the larger, and also the more important nodes of the internal jugular chain are the subdigastric, the bifurcation nodes and the supra omohyoid ones (Taylor and Nathanson[17]). The subdigastric or jugulodigastric lymph node is situated just below or partly medial to the posterior belly of the digastric muscle where this crosses the internal jugular vein. The bifurcation nodes are those lying particularly at the level of the carotid bifurcation, and therefore they consist of lower members of the upper group and upper members of the middle group. Of the supra-omohyoid nodes, the lower ones of the middle group, a particularly large one is sometimes known as the jugulo-omohyoid node since it lies about where the internal jugular vein is crossed by the omohyoid muscle. The lower nodes of the internal jugular chain, below the omohyoid muscle, frequently termed "supraclavicular nodes," are less constant and less important for they are not nearly so often involved with metastatic cancer as are the other nodes. However, for complete radical extirpation of the cervical lymphatics they most assuredly should be included in the block dissection. They are largely under cover of the sternocleidomastoid muscle and the clavicle, but it is their lateral extension that appears in the lowest part of the posterior triangle of the neck as the transverse cervical chain.

The dissection up to this point has included the nodes of the internal jugular chain up to about the level of the posterior belly of the digastric muscle. The further dissection, to be described, removes the uppermost members of the chain and the submaxillary and submental nodes.

Vascular and Nervous Relations of the Posterior Belly of the Digastric Muscle. The region of the posterior belly of the digastric, now being approached in the dissection, is one of especially complicated anatomy. Careful attention must therefore be given to dissection in this area. This belly of the muscle is perhaps one of the most important landmarks to the surgeon in this region. Superficial to it there are no vital structures. The great auricular nerve and the cervical branch of the facial nerve both have this course, but have already been severed. Also, the posterior facial or common facial vein passes across it superficially; and if it is not properly secured as the dissection advances, it may give rise to some very annoying bleeding. Deep to the posterior belly of the digastric lie the internal and external carotid arteries and the internal jugular vein, the tenth, eleventh and twelfth cranial nerves, and the sympathetic trunk.

Carotid Branches. As already noted, the superior thyroid artery commonly arises from the external carotid artery close to the origin of the latter vessel, and therefore usually below the level of the greater cornu of the hyoid bone. The lingual artery, which like the superior thyroid arises from the anterior surface of the external carotid, usually arises just below the level of the digastric at about the level of the body of the hyoid and courses forward deep to the muscle to appear in an inferior part (Lesser's triangle) of the digastric triangle (Hollinshead[10]); the lingual may arise higher, with the facial. The ascending pharyngeal artery also arises from the infradigastric portion of the external carotid but from the posteromedial surface of the vessel, hence is usually not seen in neck dissections. Just below, or under cover of, the posterior belly of the digastric, the facial or external maxillary artery typically arises from the anterior surface of the external carotid artery, while the occipital artery arises from its posterior surface. The facial and lingual arteries may arise together, sometimes in the position more typical of the lingual artery and sometimes in that more typical of the facial.

It may be noted here that branches of the external carotid which course posteriorly regularly cross the internal jugular vein superficially and therefore must be ligated and

sectioned. These branches include (1) the sternocleidomastoid branch of the superior thyroid artery which is ligated as the sternocleidomastoid is elevated, (2) the occipital artery and its sternocleidomastoid branch, and (3) sometimes the posterior auricular artery. It is only occasionally that the latter artery must be ligated; as a rule it arises sufficiently high from the external carotid to be beyond the field of dissection, or it may arise from the occipital artery and hence it need not be ligated separately. Because the facial artery is closely associated with the submaxillary gland and with the lymph nodes at the inferior border of the mandible, a segment of this artery is removed in dissection; it may be remembered that it has already been divided and ligated over the mandible. The second division and ligation take place during the dissection above the level of the hyoid bone as the submaxillary gland is being freed.

Nerves. Of the nerves in the immediate region of the posterior belly of the digastric muscle, the hypoglossal and the spinal accessory are more superficially located, usually lying between the internal jugular vein and the muscle. The vagus and the cervical sympathetic trunk are located more deeply, posteromedial to the internal jugular vein. The facial nerve, except for its cervical and marginal mandibular branches, already encountered, and for its branch to the posterior belly of the digastric and the stylohyoid, which lies above the digastric close to its origin, is largely out of the field of radical neck dissections; so, too, is the glossopharyngeal nerve since it lies both higher and more deeply than the other nerves mentioned.

At its exit from the skull the hypoglossal nerve lies both medial and slightly posterior to the internal jugular vein and the internal carotid artery. It passes laterally and downward close to the base of the skull and behind the vagus nerve, and then between the internal jugular vein and the internal carotid artery. Occasionally it passes posterior to the internal jugular vein. As it emerges from its deeper position, it lies under the posterior belly of the digastric; and as it descends beneath the lower border of this muscle, it usually lies on the lateral surface of the internal carotid artery. In this position it descends a little bit below the angle of the mandible, gives off its descendens hypoglossi to the ansa hypoglossi and then turns forward at or above the level of the greater cornu of the hyoid bone. As it makes its forward turn, it crosses superficial to the occipital artery but is in turn crossed superficially by the sternocleidomastoid branch of this artery, so that the turn of the nerve is held in position by the origin of this branch from the occipital artery. It is usually at this point that the occipital artery is ligated. As the nerve runs forward above the greater cornu of the hyoid bone, it gives off its thyrohyoid branch to the muscle of the same name and then disappears beneath the posterior belly of the digastric and the stylohyoid muscle to enter the submaxillary triangle.

The spinal accessory nerve, after emerging from the jugular foramen between the vagus nerve and the internal jugular vein, commonly turns laterally between the vein and the internal carotid artery. In 25 to 30 per cent of sides, however, it passes posterior to the internal jugular vein, and in approximately 3 per cent passes through a venous ring formed by the vein. It descends obliquely downward and backward, commonly crossing superficial to the internal jugular vein, and about 4 cm. or more below the tip of the mastoid process either enters the deep surface of the sternocleidomastoid or gives off a branch to this muscle while the main branch of the nerve runs entirely deep to the muscle. The former is far more common. In any event, before the nerve enters the sternocleidomastoid, it is closely associated with upper lymph nodes of the internal jugular chain; and as it emerges through or deep to the posterior border of the muscle, nodes of the spinal accessory chain are associated with it. It is because of these associated nodes that we insist that the spinal accessory nerve should be routinely sacrificed in radical neck dissections.

The vagus nerve descends deep to and somewhat between the internal jugular vein and the internal carotid artery, and subsequently between this vein and the common carotid artery. Its relations at and above the level of the posterior belly of the digastric are therefore similar to its relations below. Since it is closely associated with the internal jugular vein throughout the length of this vein, it is obvious that care should be taken not to include the nerve when the vein is clamped, ligated and divided. Its superior laryngeal branch arises high in the neck, from the lower end of the

nodose ganglion of the vagus, and runs downward and forward on the pharyngeal wall deep to the carotid vessels, dividing into internal and external laryngeal branches as it does so. It is especially the external laryngeal branch (to the cricothyroid muscle and sometimes a portion of the inferior pharyngeal constrictor) that is exposed in radical neck dissections. It usually lies just deep to or just above the superior thyroid artery and should be avoided whenever possible in ligating and dividing the superior thyroid vein or artery.

The cervical sympathetic trunk, ending above in the large superior cervical ganglion, lies behind the posterior layer of deep fascia of the neck immediately on the surface of the longus capitis muscle. Because of its position, it is rarely important in radical neck dissections.

Dissection of the Remainder of the Submental and Submaxillary Triangles. Dissection of both of these triangles was started at the time of the initial, horizontal incision that formed the upper part of the T. The dissection from below has now been carried up to approximately the borders of these triangles; therefore the remainder of the dissection involves cleaning out these triangles and detaching the block of tissue at its upper end. (Fig. 5.)

In our dissection of these triangles we routinely sacrifice the digastric and the stylohyoid muscles. Our decision to do so rests on the intimate association of lymph nodes and lymphatic pathways with these muscles; some of the nodes of the submental triangle lie immediately on the anterior belly of the muscle, while important efferent pathways from prevascular and retrovascular submaxillary nodes follow the external maxillary artery deep to the submaxillary gland and lie on the deep surface of the posterior belly of the digastric and stylohyoid muscles. Further, resection of the posterior belly of the digastric and of the stylohyoid muscle allows a better approach to the upper end of the internal jugular vein and yet has no increased morbidity connected with it. Since elimination of as much as possible of the tissue of the neck which contains lymph nodes and lymphatics is the express purpose of radical neck dissection, this purpose is obviously better attained when these muscles are sacrificed.

Dissection of the Submental Triangle. As the first step in this procedure, the fascia covering the mylohyoid muscle is split upward from the hyoid bone to the symphysis of the mandible, and the anterior belly of the digastric is cut from the mandible. This, the deep fascia covering the triangle, and all the connective tissue between this fascia and the mylohyoid muscle are then stripped from the muscle, the deep fascia being detached below from the hyoid bone.

Through this procedure the submental lymph nodes, which lie on the mylohyoid muscle in the fatty tissue between the muscle and the deep cervical fascia or on the anterior belly of the digastric muscle, are removed. The anterior group of deep submental nodes, consisting usually of one or two, is not very constant; but when it occurs, it is usually found in the anterior part of the triangle near the mandible. The middle group consists of lateral and medial nodes and is rather constant; the lateral nodes usually occur on the anterior belly of the digastric about halfway between the mandible and the hyoid bone, the medial group within the triangle at this same level. The posterior group of nodes, again one or two, is inconstant like the anterior group, but when present may lie either medially or laterally near the hyoid bone. The submental nodes receive their afferents from the chin, the middle part of the lower lip, the anterior part of the floor of the mouth, the tip of the tongue and the anterior part of the teeth and gums of the lower jaw. Their efferents pass posterolaterally and somewhat downward, to nodes of the submaxillary group or medial nodes of the internal jugular chain and to the more anterior subdigastric nodes of this chain (Rouvière[15]).

In removing the anterior belly of the digastric from the mylohyoid muscle, it will be found that it cannot be readily stripped backward by blunt dissection since there is no plane of cleavage between it and the underlying mylohyoid muscle. Consequently it is freed by sharp dissection. Near its attachment to the mandible the anterior belly of the digastric does not contain any vessels of consequence; but as it is freed by sharp dissection, several vessels are encountered. These are branches from the submental artery and perforating branches from the sublingual artery. In this dissection the mylohyoid muscle is disturbed as little as possible. However, this

muscle may be sacrificed if necessary to circumvent a lesion.

Dissection of the Submaxillary Triangle. When the anterior belly of the digastric is freed as far as the hyoid bone, the fascial attachment of the digastric tendon to this bone is dissected free and the stylohyoid muscle, which splits to pass on both sides of the digastric tendon, is likewise detached from the hyoid bone. These attachments lie just above the origin of the omohyoid muscle from the bone, at about the junction of the body with the greater cornu. Through this maneuver, both the boundaries and the contents of the submaxillary triangle are included in the block dissection.

The submaxillary gland is the largest component of the digastric or submaxillary triangle and not only largely fills this triangle but also overflows its edges, so as partly to overlap superficially both bellies of the digastric muscle and extend upward medial to the mandible. On its deep surface the gland lies not only against the digastric but also between the two bellies of the muscle against the mylohyoid, hyoglossus and middle constrictor of the pharynx. Elevation of the bellies of the digastric and dissection along the mylohyoid muscle therefore separate the gland from its bed. At the posterior border of the mylohyoid, however, the gland is partially subdivided into two portions, a superficial and a deep part, for a tongue-like process of the gland (called the deep process) typically extends forward with the submaxillary duct above the mylohyoid muscle. Closely associated with the gland and its duct at the posterior border of the mylohyoid muscle are the hypoglossal and lingual nerves; this border of the muscle is therefore an important landmark.

As the mylohyoid muscle is exposed by traction on the anterior belly of the digastric and its adjacent tissue, the mylohyoid nerve and artery can usually be seen descending on the superficial surface of the mylohyoid to supply this muscle and the anterior belly of the digastric. These structures (from the inferior alveolar nerve and the internal maxillary artery, respectively) descend downward and forward in a groove along the inner surface of the ramus of the mandible.

When the mylohyoid muscle is exposed to its posterior border, a ribbon retractor is placed under its posterior edge in order to expose the important structures passing deep to it, namely, the lingual nerve above, the hypoglossal nerve below and the submaxillary duct between the two nerves.

The lingual nerve is identified and separated from the submaxillary duct and surrounding tissue by buttonholing the tissue between the nerve and duct and placing a clamp just below the nerve. The ligature should include a constant vessel which is found in this location, and also the branches from the submaxillary ganglion which go to the gland. The submaxillary ganglion is suspended from the lingual nerve as this nerve turns forward to pass deep to the mylohyoid and lies on the hyoglossus muscle or the lower border of the styloglossus muscle, as these muscles intersect at their entrance into the tongue. The ganglion is sometimes mistaken for a lymph node and dissected, but it should remain undisturbed if possible. It receives its preganglionic fibers through the chorda tympani, which joins the lingual nerve high in the infratemporal fossa shortly after this nerve separates from the inferior alveolar.

After the lingual nerve has been safely separated from the submaxillary duct, two clamps are applied to this duct (care being taken that they are on the duct alone, since the lingual nerve in its further course to the tongue loops around the duct), which is divided and ligated close to the anterior clamp. The posterior clamp is retracted backward as a part of the block specimen.

The hypoglossal nerve is then sought in order to avoid its accidental transection posteriorly. As it disappears above the posterior border of the mylohyoid muscle, it lies deep to the submaxillary duct and the accompanying deep process of the gland immediately on the hyoglossus muscle. It is accompanied in this course by the vena comitans of the hypoglossal nerve, the chief venous drainage of the tongue. Since it supplies all of the musculature of the tongue, its transection produces unilateral atrophy of the tongue.

From the posterior border of the mylohyoid muscle the hypoglossal nerve can be traced backward in its course through the submaxillary triangle across the hyoglossus muscle. At the posterior border of this muscle the lingual artery lies deep to the nerve (this artery passes on the deep surface of the hyoglossus, while the nerve runs on the superficial surface

of the muscle), and it is here that the hypoglossal nerve and the two bellies of the digastric muscle can be made to form a small triangle, Lesser's triangle, which is the classic area for ligation of the lingual artery. Actually, the lingual artery may lie either above or below the level of the nerve, but the hypoglossal nerve can always be pushed up sufficiently to include the artery in the triangle. Posterior to Lesser's triangle the nerve lies beneath the posterior belly of the digastric. It is at about the lower posterior border of this muscle that it makes its turn forward around the occipital artery and its sternocleidomastoid branch. The occipital artery is usually ligated and divided at the point at which this nerve turns around it. If there has been a previous laryngectomy, and especially if the hyoid bone has been sacrificed, this nerve may be bound down by considerable scar tissue which makes identification of it difficult.

As the submaxillary gland with the tissue associated with it (except for the nerves which have already been freed) is loosened from its bed, it will be found to be still held on its deep surface by the external maxillary or facial artery. This artery has already been ligated and sectioned above the upper border of the mandible, but is ligated and sectioned once again deep to the submaxillary gland in order to allow removal of this gland and its associated lymph nodes. As already noted, the external maxillary arises from the anterior surface of the external carotid, either in common with or above the origin of the lingual artery and, variably, at about the lower border of the posterior belly of the digastric deep to or above the muscle. Unless its origin is high, this artery typically loops upward and may in this part of its course come in close contact with the posterolateral wall of the pharynx in the tonsillar region. Here it gives off its tonsillar branch as well as an ascending palatine branch and then turns downward once again. In so doing it comes in close contact with the deep surface of the submaxillary gland and lies in a groove on this gland, or may indeed be completely surrounded by glandular tissue. It gives off into the submaxillary gland a number of branches; and as it emerges superficially between the gland and the inferior border of the mandible, it sends a submental branch forward into the anterior part of the digastric triangle and on into the submental triangle.

Branches of the submental branch of the facial artery are routinely observed and ligated if necessary during neck dissection, and so are the glandular branches. The tonsillar and palatine branches of the artery are seldom seen in the course of this dissection because of their higher origins. Once the glandular branches of the artery have been cut and sectioned or the stem of the artery before it enters the gland has been divided, the gland can be dissected by the finger from its deep and posterior attachments and then retracted as a part of the specimen.

Freeing the Block Posterosuperiorly. Attention is then transferred to the upper and posterior portion of the block where the sternocleidomastoid muscle and the posterior belly of the digastric are detached from the mastoid process by scissor dissection. (Fig. 6.) The sternocleidomastoid muscle inserts by a tendon into the lateral surface of the mastoid process from its tip to its superior border, and by a thin aponeurosis into the lateral half of the superior nuchal line of the occipital bone. The posterior belly of the digastric takes origin from the medial side of the mastoid process at the mastoid notch. Just medial to the origin of the digastric, the occipital artery passes backward.

In detaching these muscles the specimen is put on anterior traction as the sternocleidomastoid is cut free, close to the mastoid process. Bleeding vessels are always encountered here and should be clamped as the muscle is being freed from the bone. They are mainly branches of the posterior auricular and occipital arteries and veins. After the sternocleidomastoid is freed, the digastric is detached from the medial aspect of the mastoid process. Finally the stylohyoid muscle, which has already been detached from its insertion into the hyoid bone, is transected at its origin on the styloid process. The styloid process lies anterior and deep to the mastoid process and gives attachments to the stylohyoid, styloglossus and stylopharyngeus muscles, in addition to stylomandibular and stylohyoid ligaments. The main stem of the facial nerve passes superficial to the styloid process as the nerve runs forward to enter the parotid gland.

The stylohyoid is the most superficial of the three muscles attached to the styloid process. It arises from the posterior and lateral surface of the process and extends diagonally down-

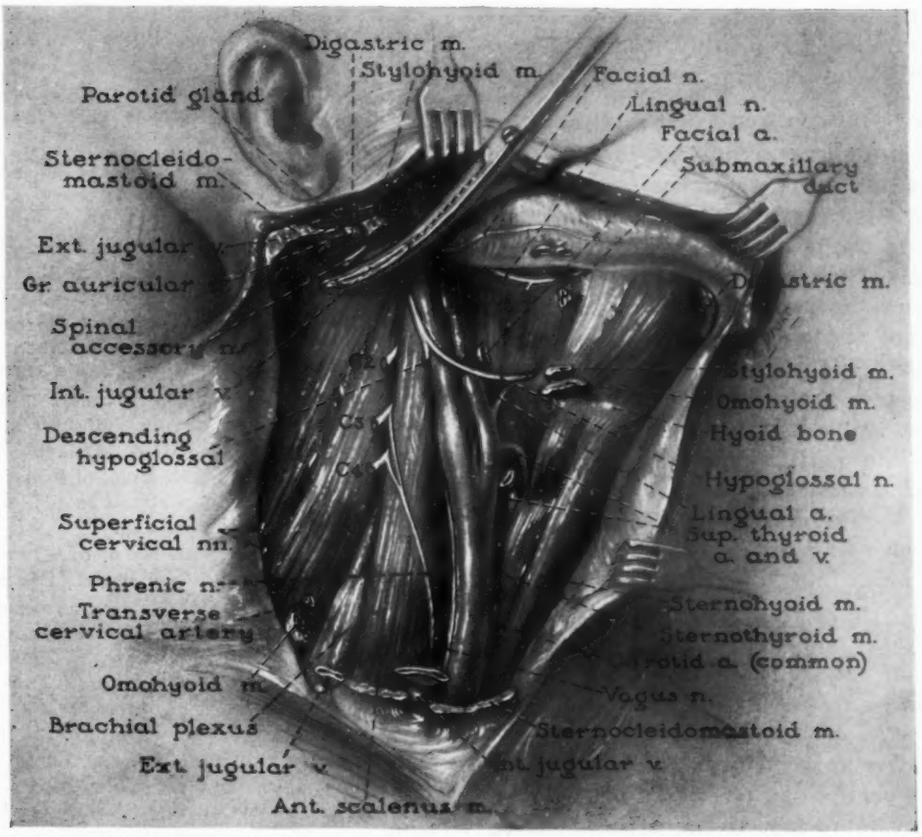

FIG. 6. The dissection has been completed and the sacrificed tissues have been removed. The internal jugular vein is divided and ligated as close to the jugular foramen as possible. Any of the remaining structures may be excised in whole or in part if such excision will circumvent the malignant growth.

ward, forward and laterally close to the posterior belly of the digastric, usually splitting to pass both superficial and deep to the digastric tendon before inserting on the hyoid bone. The styloglossus, a shorter muscle, arises from the lateral and anterior surface of the process usually close to its tip; and since it goes to the tongue, it is directed more forward than is the stylohyoid. The stylopharyngeus muscle lies more deeply, arising from the medial surface of the styloid process, and the stylohyoid ligament arises from the tip of the process. Since the external carotid artery passes deep to the posterior belly of the digastric and to the stylohyoid muscle but emerges between the stylohyoid and the styloglossus muscles to lie more superficially, care must be exercised in this region if severe hemorrhage from the external carotid artery is to be avoided. If necessary to circumvent the lesion, the external carotid artery may be sacrificed.

The specimen is put on alternate anterior, upward and posterior traction as the tissues binding it down in this area are freed. Once the muscles and blood vessels entering the block are detached, the further attachments are largely areolar except for the accessory nerve and the internal jugular vein itself. The nerve is identified as it lies on or close to the internal jugular vein and is sectioned as high as possible. Since it has already been sectioned as it passed beneath the anterior border of the trapezius, a large portion of this nerve is included in the block.

Following transection of the spinal accessory nerve, only the internal jugular vein should be holding down the specimen. Two curved clamps are applied as high up on this vein as possible, with their convexities pointing upward, and a third clamp is applied about ½ inch (approximately 1.3 cm.) or so below these two. The vein is then cut just below the second clamp and the specimen is freed. (Fig.

6.) The vein is doubly tied, thus completing the dissection except possibly for a few additional bleeding vessels which are also tied off.

Drainage, Suturing and Postoperative Care

After hemostasis is assured, two large Penrose drains are inserted for the whole length of the operative field. One extends from the upper part of the posterior region down to the lower limits of the dissection, and the other extends from the symphysis to the angle of the mandible and then vertically downward. Both drains are brought out through a stab incision posterior to the operative incision. This allows for better healing of the lower portion of the vertical incision than would occur if the drains were carried out through this part of the incision. The lower ends of the drains are sutured to the skin. The drains are removed on the fourth or fifth postoperative day.

The incisions are closed with interrupted sutures, plain catgut being employed for the platysma and its associated fascia, and black silk for the skin.

Postoperative care includes the intravenous use of fluids on the day of operation and possibly the day following. If the dissection was a combined one, removing not only the metastatic neck lesion but also the primary one, a feeding tube is of course placed in the stomach preoperatively and feedings are instituted the day following operation. Antibiotics are routinely used in combined dissections, but in radical neck dissections alone they are usually not required. Early ambulation is encouraged, the patient usually being urged to start moving about the day following operation. Hospitalization is usually five to seven days postoperatively.

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Treatment of the Flail Chest by Towel Clip Traction

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THE flail chest is one of the true emergencies in traumatic surgery since mismanagement may lead to a fatal consequence. There are many diversified views as to the proper management of these cases and little knowledge as to the technic of rib traction.

The recent literature discloses little detail regarding the management of the flail chest in spite of frequent mention of the importance of the correction of paradoxic respiration. Some authors mention the use of padding followed by adhesive strapping.[5] Others mention traction,[2] but give no detail as to technic. Sweet[6] states that "in such cases open exposure and fixation of the fractured rib ends by wire sutures is a life-saving measure." This, however, is a formidable procedure. Johnson and Kirby[4] recommend immobilization—pad and strap and apply a sand bag or better "free a small rib segment subperiostally through a small incision under local anesthesia, and then pass a stainless steel wire around it. A weight of five pounds is attached to a rope and suspended over a pulley. Towel clips and specially designed clamps may also be used for applying traction to the thoracic cage." In discussing the treatment of the severe flail chest a report from the Fitzsimmons Army Hospital[7] mentions applying a towel clip around the central rib of a "flail" segment and applying traction, usually over five pounds, suspended over a single pulley attached to an overhead frame. Some articles[1] mention the use of traction to the sternum in cases of fractured sternum and "stove-in" chest. Jaslow[3] mentioned its possible use on the ribs.

CASE REPORT

Patient W. B., a twenty-eight year old sailor, was admitted unconscious to the Aberdeen Proving Ground Hospital on December 8, 1953, following an automobile accident. Blood pressure was 100/65, pulse 92 and respirations 38 (gasping in character). There was an odor of alcohol on the patient's breath and multiple lacerations on his face. The pupils were small but reacted to light, and the eyes were roving. The right chest sounded clear and no breath sounds were heard on the left. Light palpation revealed free mobility of left rib cage. Paradoxic respiration was noted on the left; and over the left chest and abdomen a marked amount of edema and subcutaneous emphysema were present. The abdomen was flat with spasm of the muscles of the left upper quadrant. Rectal examination was negative. Eight hundred cc. of clear urine were removed by catheterization and the catheter was left in place. All extremities were felt to be intact. Reflexes were diminished throughout; Babinski sign was negative. Blood was drawn for studies and cross-matching, and intravenous therapy was started.

The patient was x-rayed, portable oxygen being administered. Films revealed no fracture or dislocation of the cervical vertebra. X-ray of the chest (Fig. 1) showed the following on the left: fractures of ribs 1 through 11, with multiple fractures of ribs 4 through 10. There was marked over-riding of many of the fragments. The left chest was "stove-in." There was a density in the left lower lung field. This had the appearance of interparenchymal hemorrhage. Marked subcutaneous emphysema did not permit proper evaluation of the pneumo-

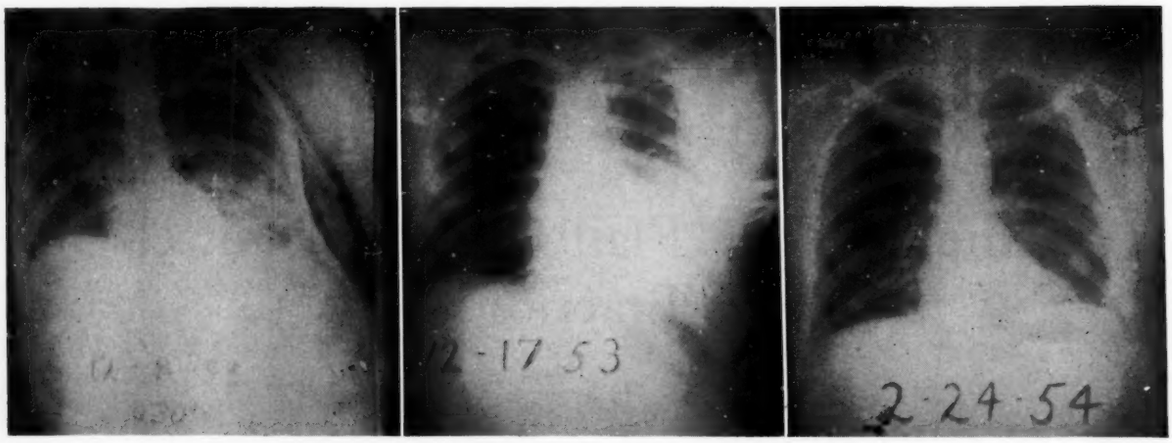

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FIG. 1. Admission x-ray of the chest showing "stove-in" deformity and subcutaneous emphysema.

FIG. 2. After nine days' traction the "stove-in" deformity is corrected and the ribs appear to be well aligned. Subcutaneous emphysema is gone.

FIG. 3. Discharge film. There is normal chest contour. Callus is present. Lung fields are clear.

thorax. Fractures of the transverse processes of L1 through L4 were noted on the left. Renal shadows could be seen and appeared normal. Towel clip traction was applied to left ribs 5 and 8 in the mid-axillary line with five pounds' weight to equalize the paradoxic respiration. Nasal oxygen was started. A needle was placed in the second anterior interspace on the left and connected by tubing to an underwater seal. Air started bubbling immediately. Penicillin-streptomycin and tetanus toxoid were administered.

Laboratory data were as follows: hemoglobin, 14.2 gm.; red blood cells, 5,150,000; white blood cells, 14,500, with polymorphonuclears 68 per cent, lymphocytes 31 per cent and monocytes 1 per cent. Urinalysis showed the following: clear urine with an acid reaction; specific gravity, 1.001; albumin, 1+, with microscopic appearance of 4 to 6 white blood cells per high-power field, occasional epithelial cells and 1 to 3 red blood cells per high power field.

Three hours later blood pressure was 90/40, pulse 112 and respirations 28, being smooth and even. Temperature was 37.5°C. rectally. The patient remained unconscious throughout these procedures. The tube from the chest needle was bubbling air under water. At this time breath sounds were noted anteriorally on the left; none were noted posteriorally in the left lower chest.

Four hours later physical signs were unchanged; the hemogram also was unchanged. Urinalysis showed an increase in the number of red cells. There was little urinary drainage. Non-protein nitrogen was 31 mg. per cent. Since no urinary drainage occurred, a cystogram was done on the evening of admission. The bladder filled and emptied normally. External pressure appeared to be present on the superior aspect on the left. This was thought to represent a retroperitoneal hematoma. Intravenous fluids were then restricted to minimal requirements plus loss. Electrocardiogram revealed sinus tachycardia.

On the following morning, the patient was semi-conscious. Blood pressure was 160/100 and the pulse 112; respirations were 24, of even character. Lungs were clear except for decreased percussion and breath sounds at the left base, with ronchi in the left mid-lung field posteriorally. Air was still bubbling from the chest catheter. The abdomen was soft, and peristalsis were now present. During the previous evening 40 cc. of urine had been passed. Eye grounds appeared normal. The Babinski sign was now positive on the left. Blood non-protein nitrogen was 81 mg. per cent; CO_2, 44.3 volumes per cent; and blood chloride, 103 mEq./L. Urinalysis showed marked increase in red and white blood counts, and a 4+ albumin. Hematocrit dropped to 36 and the patient was given 1,000 cc. of blood slowly. Portable x-ray film of the chest showed that the lower rib cage had been pulled out and that the upper towel clip had pulled off the rib. However, the chest wall was being pulled out by the weights. Later the towel clips were reapplied on ribs

4 and 5 in the anterior axillary line. The subcutaneous emphysema had not progressed and the density in the left lower lung field was unchanged.

On December 10th physical examination was unchanged. The urinary output was increased and fluids increased accordingly. Babinski sign was positive on the left. The patient was able to move all extremities well. Blood pressure was 180/100. The chest catheter had stopped draining air. It was left in place another twenty-four hours and was removed when no further drainage occurred.

The following day a Levin tube was inserted into the patient's stomach for feeding purposes. His state of consciousness slowly improved. Urinary output remained good. Lungs were otherwise unchanged. The patient was given 500 cc. of blood on December 14th because of another drop in the hematocrit. The subcutaneous emphysema had gradually subsided but there was still a marked amount of edema of the left chest wall. Admission blood alcohol was reported as 1.9 mg. per cent. Levin tube feedings were continued for five days. At that time the patient was alert enough to take all feedings.

Portable x-ray of the chest on December 17th (Fig. 2) showed better alignment of the fractured ribs, correction of the "stove-in" deformity and persistence of the density in the left lower field. On December 21st, the nonprotein nitrogen was 36 mg. per cent; the chest wall was now stable enough to permit removal of the towel clips. At the site of one of the clips a slight area of necrosis was present which had been caused by pressure of the skin and subcutaneous tissues between the blades in the towel clip. A sterile dressing was placed over this. Deep breathing exercises were begun, and the patient was permitted to sit in a wheel chair. His blood pressure was now 130/100 and became normal thereafter.

On December 23rd the patient was transferred to the Bainbridge Naval Hospital. A thorough radiologic examination was performed which showed a fracture of the left scapula in the region of the acromion with the body in good position, a moderate left acromioclavicular separation, previously named fractures of ribs 1 through 11 on the left side, with three fractures in ribs 5 through 8; also a density in the left lower field of the lung and a fracture of the left lumbar transverse processes. No skull fracture was present. The acromioclavicular separation was treated by the orthopedic department. Hemogram was normal. Urinalysis showed only a trace of albumin and 0 to 3 red blood cells per high-powered field. Blood urea nitrogen was 25.6 mg. per cent. A few days later incision and drainage were performed for an abscess which had developed at the site of necrosis caused by the towel clip.

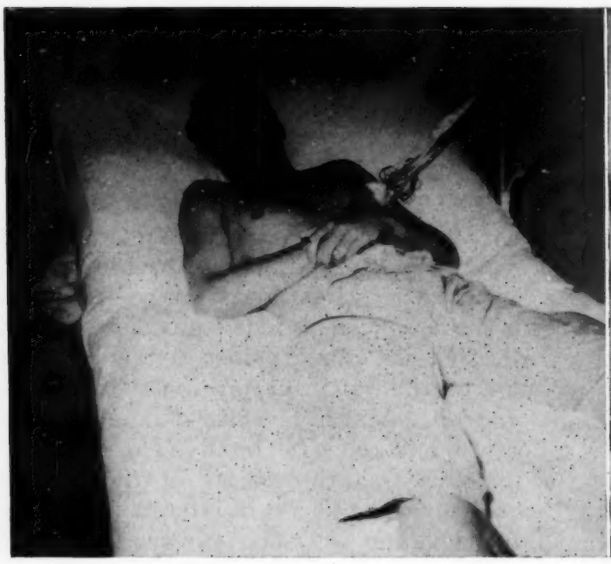
Intravenous pyelogram showed no function on the left. Thereafter, cystoscopic examination revealed a normal bladder, normal dye excretion on the right and none on the left. Retrograde pyelogram revealed normal appearing calyces and pelvis bilaterally. The presumptive diagnosis was thrombosis of the left renal artery or vein. This is of interest in relation to the elevation in blood pressure and anuria which occurred shortly after admission, however, the blood pressure soon dropped to normal and remained so, as did the urinary output. Follow-up intravenous pyelogram six weeks later revealed no function on the left.

By January 22, 1954, x-ray of the chest revealed complete clearing of the parenchymal changes at the left base. Some pleural thickening was present. Formation of callus was present at a number of the rib fracture sites. On February 24th (Fig. 3) x-ray of the chest revealed complete resolution of the parenchymal changes in the left lung base and the various rib fractures appeared to be healing quite satisfactorily. Pleural thickening was minimal. Physical examination of the chest revealed clear lung fields. Follow-up laboratory studies continued to show a few red cells per high-powered field and a trace of albumin in the urine. On January 5, 1954, the blood urea nitrogen was 29 mg. per cent and on March 3rd it was 20.6 mg. per cent.

The patient was discharged in March, 1954. He is now doing full duty and will be followed by the Urologic Service at Bainbridge Naval Hospital.

OBSERVATIONS

The previous sequence of x-rays show the gross anatomic changes involved. However, correction of the pathologic physiology is the more urgent factor. Maintenance of the negative intrapleural pressure, re-establishment of the elastic continuity of the chest wall, correction of paradoxic respiration and mediastinal motion are the main principles involved. This



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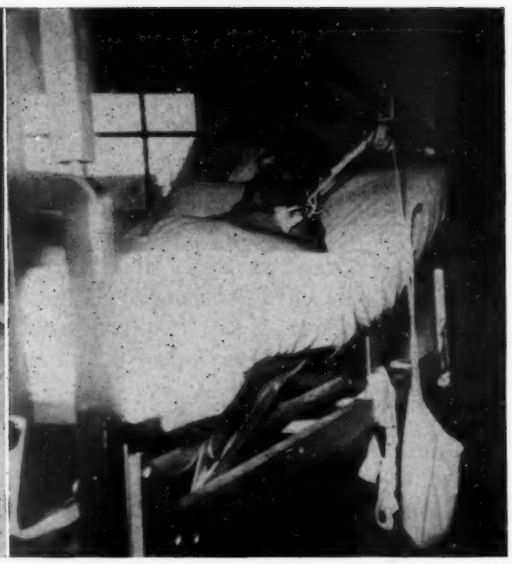

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FIGS. 4 and 5. Towel clip rib traction; note that pulley can be moved.

paper will not go into any detail on pulmonary physiology. The aforementioned treatment of flail chest by padding of the area followed by adhesive strapping will correct paradoxic respiration and mediastinal motion. However, that portion of the lung is compressed. A patient with most of one side of his chest "stove-in" would have one lung inactivated by such therapy, not to mention the chance of pulmonary puncture by a loose rib fragment. This therapy, I am sure, is adequate for a localized area involving a few ribs with little or no physiologic imbalance. For anything larger, I believe traction is the proper method.

I have found the following technic to be rapid and effective. On occasion, the towel clips have been applied in the admitting dispensary and the chest wall held out by manual traction. The skin is prepared in the usual manner and the area draped with sterile towels. Gloves are worn. The proper site is usually the central portion of the depressed area. I have chosen to use two towel clips to equalize the traction, i.e., ribs 4 and 6 for fracture of ribs 2 through 8 or more. The direction of traction is naturally to correct the deformity. The skin, subcutaneous tissues and periosteum of the rib are infiltrated with procaine. A large Backhaus towel clip is opened. The upper point is inserted just above the selected rib. It is inserted in a hooking fashion until it meets the upper inner border of the rib. The handles are then brought together closing the points on the rib. It sometimes facilitates entry of the lower point by making a small skin incision at its site of entry. The Fitzsimmons report[7] mentioned having the towel clip completely encircle the rib. This is adequate and does avoid one complication, that is, pulling away from the rib of the clip. Even when the clip does pull away, the elastic chest wall is still pulled outward by the traction. With the encirclement of the rib there is the danger of injury to intercostal nerve, artery or vein by pressure, or of pulmonary laceration and its attendant complications. By means of a rope, the two clips are attached to a five-pound weight, the rope going over a pulley at the side of the bed. (Figs. 4 and 5.) The level of the pulley is consistent with the direction of the traction. The discomfort from the clips is no more than that from the fractures.

With ordinary movements in bed there is no need to alter the traction. The chest wall becomes stable in two to three weeks depending on the degree of the pathologic condition. In some cases the costal margin is fractured. This can be sutured with the patient in bed, using local anesthesia when the patient's condition is stabilized. This adds marked stability to the chest wall and hastens the time for removal of the clips. Stainless steel wire was used at first. However, with constant motion this seemed to cut through. Therefore, silk sutures have been used. The traction in no respect interferes with proper therapy of hemothorax or pneumotho-

rax. If the patient is moved far forward in bed, the pulley can likewise be moved forward. In all cases the patients have been able to eliminate their own secretions, but tracheal aspiration and tracheotomy are to be considered when indicated.

Complications of this therapy are few. Necrosis of the skin and subcutaneous tissues between the blades of the towel clip was occasioned in this case by the severe edema of the chest wall. This might have been avoided by either not closing the catch on the handles and depending upon the traction to hold the clip in place, or by using a single hooked instrument. In this case an abscess did develop; however, it was easily handled by local therapy. In an unreported case there was a draining sinus from one rib that had had traction applied to it. In this case specific antibiotic therapy was instituted after sensitivity studies. Injury to the intercostal vessels has never occurred. Also, movement of the rib fragment too far away from the chest wall has not occurred.

It should be remembered that associated injuries are almost always present. Clavicular and scapular injuries were usually present. Cerebral and intra-abdominal injuries must be carefully watched for. Associated fractures of the long bones have not been seen.

With the automobile becoming a projectile of destruction, crushing injuries to the chest will appear more frequently, and the surgeon will be increasingly confronted with the problem of the "flail chest."

SUMMARY

1. Recent literature in regard to the "flail chest" is discussed.
2. A case of "flail chest" treated by towel clip traction is reported.
3. The technic of towel clip traction with its attendant complications is reviewed.

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Basal Cell Carcinoma Occurring in a Fistula-in-Ano*

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BASAL cell carcinoma of the anus is an extremely uncommon lesion and is therefore of great interest, particularly to the proctologist, A careful survey of the literature reveals that only eleven cases have been reported. In 1933 Buie and Brust[1] reviewed 2,939 malignancies of the anus and rectum at the Mayo Clinic. Fifty-one of these were primary anal neoplasms and only two were basal cell tumors. In 1935 Guess[2] studied 150,000 biopsy reports filed at the state institute for malignant disease at Buffalo, New York, and found no basal cell carcinoma of the anus. The literature dealing with basal cell tumors of the anus is scanty, and in an era of rapidly increasing cancer consciousness among laymen and physi-

TABLE 1

Author*	Age (yr.) and Sex of Patient	Clinical Findings	Treatment	Result
Tucker and Hellwig, 1938[5]	43 M	Early lesion; less than 2 cm. origin in hair follicle	Local excision and radium	No recurrence 3 yr. after surgery
Guess, 1935[2]	52 M	Early lesion, 2 cm.	Local excision, radon seeds	No recurrence 5 yr. later; died of other causes
Gabriel, 1946[4]	70 M	Early 1–2 cm. lesion; biopsy confirmed diagnosis	Excision by cutting diathermy	No recurrence after 1 yr.
Buie and Brust, 1933[1]	53 F	Advanced lesion, size of orange; mass left groin	Radium advised; went home without therapy	Unknown
Buie and Brust, 1933[1]	53 F	Advanced annular lesion	Colostomy, posterior resection; half of vagina and local nodes removed	Recurrence in 8 mo.; death in 9 mo.
Lawrence, 1941[6]	73 M	Advanced annular lesion, fungating, invading prostate and urethra	Loop colostomy, posterior resection	Death in 2 yr.
Lawrence, 1941[6]	73 M	Advanced annular lesion, fungating, involving anus and perianal tissues	Colostomy, posterior resection	Died postoperatively; no autopsy
Lott and Alexander, 1949[7]	62 F	2 by 1 cm. anal lesion	Local excision and x-ray	No recurrence after 7 mo.
Gross, 1948[11]	49 M	2 cm. ulcer anterior to anus	Local excision	No recurrence after 2 yr.
† Gross, 1951[12]	71 M	Anal lesion	Local excision	No recurrence after 2 yr.

* One case was recorded by Rosser in 1934.[8]
† Personal communication.

* Read before the New York Proctologic Society, October 8, 1953.

cians it is unlikely that many lesions of this type have passed unnoticed.

Basal cell carcinoma may occur at any age but is uncommon in childhood and in adults under the age of 40.[3] They are found most often as solitary tumors, but multiple lesions also occur. Characteristically, this neoplasm may appear as a superficial or deep nodule or as an indurated ulcer and may or may not be pigmented. Basal cell cancers rarely develop primarily on mucosal surfaces but may be located there as a result of secondary extension. Usually they are not highly malignant tumors but grow slowly and metastasize infrequently.

Table 1 summarizes the eleven previously reported cases of basal cell carcinoma of the anus.

We have recently completed a three-year follow-up study of a patient with basal cell carcinoma of the anus which developed in a fistulous tract. We present this patient as the twelfth reported case.

CASE REPORT

O. W. was a seventy-two year old white man with a fistula-in-ano of twenty years' duration. During this period of time the patient was rarely free from perianal drainage. He was treated surgically thirteen years before but the fistula recurred at the same site one year later. We first saw the patient four years before, when he consulted us for treatment. He was a known diabetic, controlled by diet alone.

Physical examination was essentially normal except for the proctologic findings. On the right anterolateral aspect of the anus there was a bluish swelling the size of a nickel with the external opening of a fistula in its center. Sigmoidoscopic examination was negative 25 cm. No definite internal opening of the fistulous tract could be found prior to operation. No lymph nodes were palpable in either groin.

At operation, under spinal anesthesia, the bluish lesion was excised widely in continuity with the fistulous tract. The fistula extended into the anal canal and the internal opening was found radially opposite the external opening at the crypt level. In excising the fistula, the subcutaneous and part of the superficial external sphincter muscles were divided. The wound was then packed with oxycel® gauze and a pressure dressing applied.

The postoperative course was uneventful and the patient was discharged from the hospital on the fourth day after surgery.

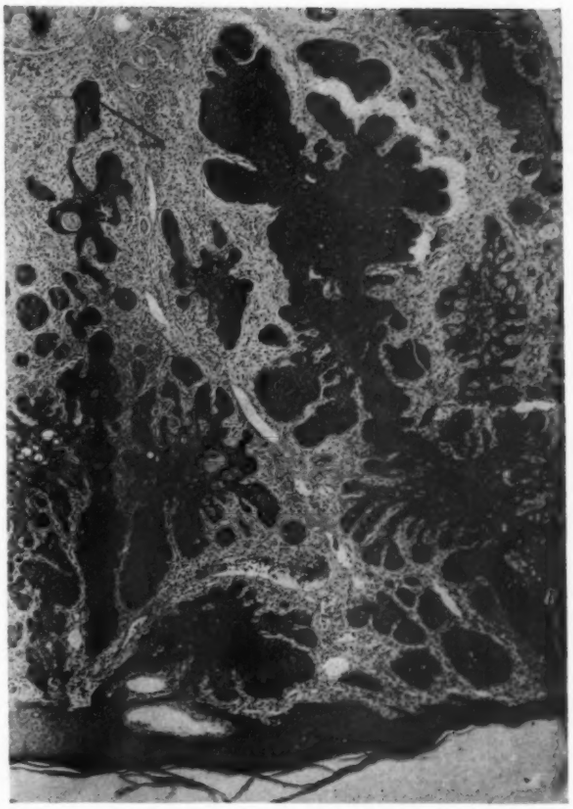

FIG. 1. Classical areas of basal cell carcinoma of the anus (low power).

Pathologic examination of the gross specimen revealed one large mass of epithelial and subepithelial tissue measuring 9 by 3 by 2.5 cm. At one end of the specimen there was a nickel-sized, dark bluish discoloration with considerable induration. A subepithelial sinus tract was present. Microscopically the surface epithelium showed focal destruction due to underlying inflammation. In the derma there were large groups of small, deeply stained cells arranged in compact masses. There were columnar-shaped cells with scanty cytoplasm and small dark nuclei resembling the basal cells in the retepegs, which grew downward into the derma but did not extend beyond. Occasional large atypical cells were present and contained mitotic figures. In addition to this there were large collections of pigment scattered throughout the tumor. Between the tumor cells the connective tissue was densely packed. No prickle cells were seen and the cells maintained a classical basal cell appearance throughout. The diagnosis was pigmented basal cell

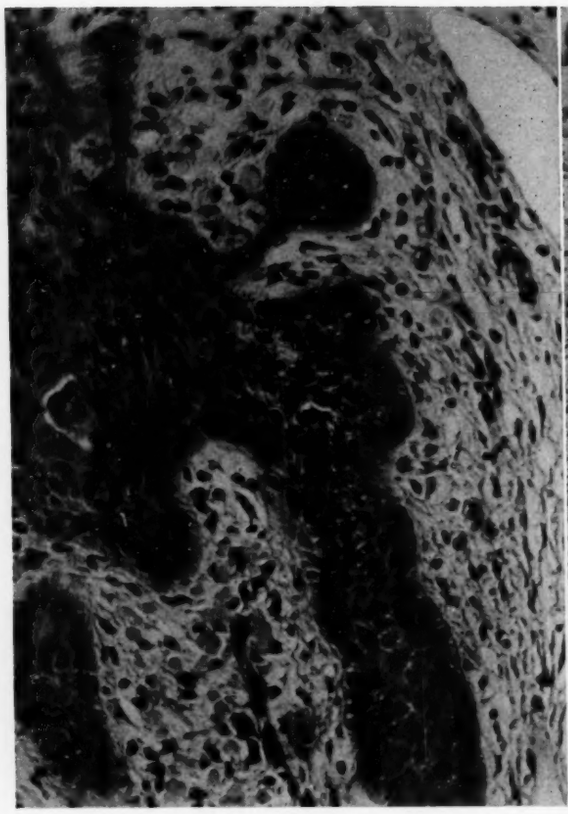

FIG. 2. Atypical basal cells (high power).

FIG. 3. Pigmented areas of basal cell carcinoma (medium power).

carcinoma showing moderate secondary inflammation. (Figs. 1 to 3.)

A radiotherapist was consulted concerning the advisability of postoperative radiation. Because we believed that the lesion was widely excised, no x-ray treatment was administered.

The patient returned for periodic follow-up examinations, and when last seen four years after operation there was no evidence of recurrence.

COMMENT

The theory that a malignant lesion may develop from a chronic, benign inflammatory condition over a period of several years has been discussed repeatedly and need not be elaborated upon further. Doran[9] has emphasized that one should not consider a fistula as having undergone malignant degeneration unless it is certain that no tumor exists in the anal canal or rectum and that the internal opening does not arise in malignant tissue. Rosser[10] has set forth three criteria for determining whether an anal fistula has undergone malignant degeneration: (1) The fistula must have existed long enough to exclude the possibility that the carcinoma antedated the fistula. (2) The tumor should not be present in the anal canal or rectum unless by secondary extension. (3) The primary or internal opening should be in an anal crypt or ulcer and not in malignant tissue.

The case which we have presented fulfills all of these criteria, and we believe that the basal cell carcinoma developed as a result of the long-standing anal fistula and the associated chronic inflammatory reaction.

The treatment of choice in basal cell tumors of the anus can be surgical, but x-ray therapy gives equally good results.

We wish to emphasize that routine microscopic examination should be made of all tissue removed at anorectal operations whether or not there is any clinical suspicion of malignant disease. The basal cell lesions reported by Guess, and Tucker and Hellwig were found because of such routine studies. The latter authors[5] reviewed 951 anorectal specimens removed at operation and found a 1.9 per cent incidence of malignancy where none was sus-

pected at surgery. Many so-called minor anorectal operations are performed throughout the country at small institutions which may or may not have adequate facilities for pathologic examination. The surgeon should be cognizant of the fact that a neoplasm may pass undetected unless he insists upon microscopic examination of all operative specimens.

SUMMARY

1. The literature on basal cell carcinoma of the anus is reviewed and eleven previously reported cases are presented.

2. An additional case of basal cell carcinoma of the anus is reported.

3. The relationship of fistula-in-ano and carcinoma is discussed.

4. The need for routine microscopic examination of all tissue removed at surgery is emphasized.

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Venous Thrombosis of the Leg Stump

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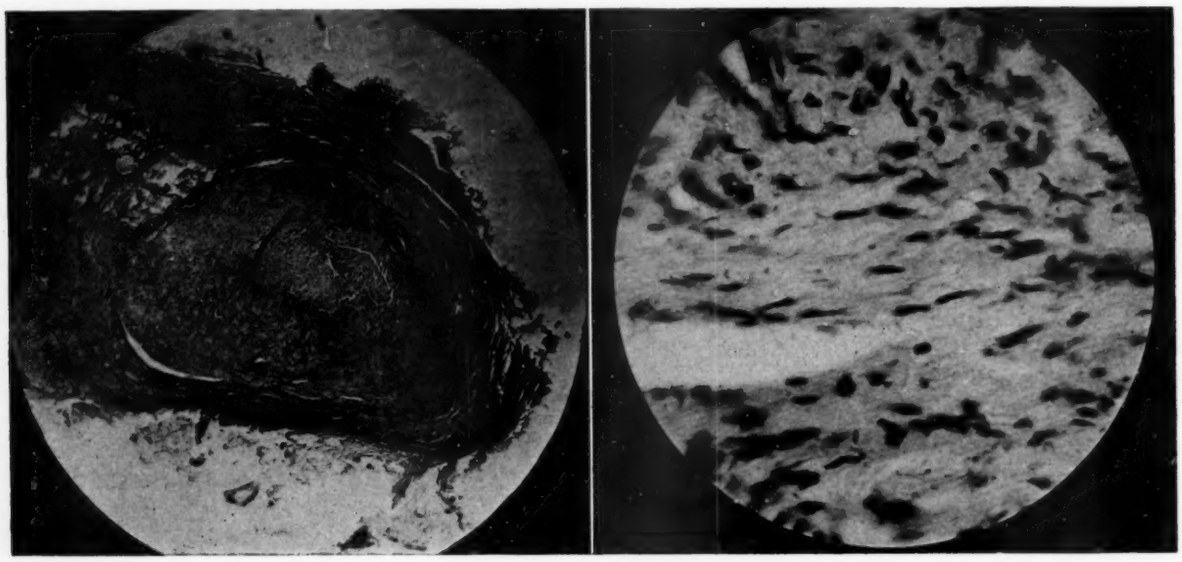
THE amputee who has lost one or more lower extremities is subject to many complications of an otherwise perhaps efficiently functioning stump and prosthesis. This fact is not startling. No stump except the supramalleolar and supracondylar has any tissues (knee and head) that have ever borne weight. Amputees with stumps other than these walk on tissues that must be conditioned to weight bearing in only one lifetime as compared with the countless generations that have prepared the sole and heel. No stump offers as much weight-bearing surface as the sole and heel combined.

The continued or acute trauma of ambulation to a stump can cause the formation of adventitial bursae. Such trauma may also blister and ulcerate the skin. Constant constriction of the stump may diminish the circulation to a point where ulceration occurs on the stump end. Very painful neuromas may occur at the ends of severed nerves. A systemic diathesis toward furunculosis or acne will see the lesions multiplied on the stump skin which is subjected to pressure, heat and moisture. The bones of a below-knee stump may separate, causing severe pain on the fibula with walking. Spurs may grow from the end of a severed bone which will be painful and may pierce the skin.



1 2

FIG. 1. Photomicrograph (original magnification × 42) of cross section of fibrosed vessel. Note thickened wall but absence of surrounding fibrosis. There was no fixation to skin or other structures.

FIG. 2. High power view, lower left of center of Figure 1, revealing vessel wall and enclosed fibrous tissue.

The following case presents a new complication for the amputee that apparently has never before been reported:

On March 30, 1954, the forty-one year old patient (the author), a below-knee amputee of some eighteen years, was struck by sudden pain on the medial condyle of the tibia, an area subjected to much of his weight in the prosthesis. It felt as though a nail had been driven into the tibia, and he could walk only on crutches. When the prosthesis was removed, a diffusely swollen tender mass was seen which measured 2 by 3 inches, ½ inch thick.

Continued pressure relief for two days by crutchwalking resulted in disappearance of the swelling, thus permitting the delineation of a hard cylindric mass in the sparse subcutaneous

fat measuring ¾ by ⅛ inch. It was freely movable within a radius of ½ inch, and pained only when pinched manually or when under pressure in the prosthesis. This important weight-bearing area is cushioned in the well shrunken stump by skin, a meager layer of fat and some adventitial tissue overlaying the periosteum. The pain on ambulation was therefore similar to that experienced in the normal heel with a marble under it in the shoe.

This tumor was removed seven weeks after the acute onset. It measured 1.0 by 0.3 cm. On gross examination it was smooth, slightly hemorrhagic and quite firm. Microscopic examination revealed a slightly thickened vessel wall (Fig. 1) filled with a mass of fibrous tissue. (Fig. 2.)

Apparently the trauma of walking initiated localized thrombosis in the lumen of the vessel which was later replaced by fibrous tissue.

BILATERAL removal of the adrenal glands, followed by postoperative medication with cortisone, is a new and experimental treatment that is supposed to prolong life and comfort in the patient with advanced neoplastic disease. However, only certain patients seem benefited by this radical procedure and it is not yet definitely determined under what conditions and in which cases bilateral adrenalectomy is worth while, if at all. (*Richard A. Leonardo, M.D.*)

Traumatic Aneurysm of the Popliteal Artery Due to Osteochondroma

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TRAUMATIC aneurysms are usually due to penetrating wounds and are of arteriovenous or purely arterial origin. Review of the literature does not reveal any cases of traumatic aneurysm due to osteochondroma. The case to be reported herein is one in which the irregular rough surface of the femur cut the popliteal artery after the cartilaginous cap of an osteochondroma was pulled off by violent exercise. This laceration allowed the aneurysm to form.

1 2

FIG. 1. Lateral view of knee x-ray showing exostosis.

FIG. 2. Lateral view of knee after femoral anteriogram. Aneurysm is shown.

CASE REPORT

Patient R. G., a fourteen year old boy, was admitted to Meadowbrook Hospital, on March 25, 1954, with a history of dyschondroplasia. Many osteochondromas had been removed previously due to functional interference. The patient was not athletic and usually did very little walking. Two weeks before admission he had walked two miles and the day before admission had ridden his bicycle vigorously for two hours after which sudden pain in the left knee occurred. Within a short time the knee and lower third of the thigh were swollen. Pain developed so that the patient could not straighten his left knee.

Physical examination revealed a young adult in good condition. The patient had deformities of all extremities and on palpation multiple bony masses throughout the skeletal system were noted. The left lower extremity showed good pulses in all arteries. The hip was kept flexed 20 degrees and the knee 45 degrees. The

foot was in equinus. The superficial veins of the leg from the knee down were distended and prominent. A large swelling was present on the posteromedial aspect of the left knee. The swelling extended from the middle of the thigh anteriorly to the same area posteriorly, and from 6 inches above the knee to below the popliteal space. Maximum swelling was in the center of the above area. There was no pulsation or bruit. The pulse could be heard with a stethoscope, as in taking the blood pressure. X-rays showed many osteochondromas. On the posteromedial aspect of the left femur at about the level of the adductor tubercle the bone was rough and irregular. (Fig. 1.)

The question of diagnosis of aneurysm of popliteal vessel or of sarcomatous changes in an osteochondroma was considered. An arteriogram of the femoral artery was done by direct vision, under anesthesia. The dye shadow showed spilling in the tumefaction and narrowing of the main arterial channel, with few collaterals present. (Figs. 2 and 3.)

Immobilization was performed with plaster. A few weeks' period of observation was decided upon to allow development of better collateral circulation.

On the fifteenth day after admission the patient complained of severe pain in the leg. The plaster was removed and more swelling was noted. All pulses were still present. The next morning the patient was awakened by excruciating leg pain. Inspection showed greater swelling of the leg and no pulses in the foot which was cool and blanched. The sudden increase of hemorrhage in the popliteal area had obliterated circulation to the foot.

Operation was performed as an emergency procedure.* Under pentothal®-gas-oxygen anesthesia a long incision was made in the upper third of the thigh and the femoral vessels and nerves were isolated. Tape was placed about the vessel to control bleeding, as required. The incision was then carried down the thigh, across the knee posteromedially to the upper third of the leg. The vessels were dissected down to the tumefaction which was a massive blood clot estimated to contain approximately 1,000 to 1,500 cc. of blood. All the tissue below the center of the mass was very edematous and almost avascular. The femoral artery

FIG. 3. Anteroposterior view of Figure 2.

was clamped off. Removal of the clot was attempted when bleeding became profuse. The circular tourniquet on the thigh was tightened and bleeding was completely controlled. The massive clot was then removed. The clot was adherent to the artery, vein and all surrounding tissues. During this phase some of the adventitia was stripped from the artery which was then dissected free down to its bifurcation and below for 2 inches. The semi-membranous and semitendinous tendons were completely freed at attachments to the tibia. A portion of the medial head of the gastrocnemius was freed from the femur. The tourniquet about the thigh was removed and was followed by rapid bleeding from the wound. The femoral artery was clamped off and then slowly released. One longitudinal tear was found in the popliteal artery. This was repaired with No. ooooo arterial silk with continuous suture and one interrupted suture. The artery was then completely released, with no bleeding. Pulse returned to the popliteal artery below the repair and also to the anterior and posterior tibial arteries.

At the site of the tear in the popliteal artery a sharp irregular area of exposed femur was noted. The rent matched with the longest of the bony spicules present. This rough area was the site of avulsion of a cartilaginous cap from an osteochondroma. The rough area together

* Surgery was performed by the author, with the help of Dr. J. Ostrowski and Dr. A. Haag.

with several smaller osteochondromas in the operative area were removed with a chisel. After ten minutes there was still no bleeding from the artery and pulse was present below the suture. The semi-membranous muscle was brought through the popliteal space and attached to the femur below the artery to protect it. The wound was closed and a long-leg plaster applied. At the end of the operation no pulse was felt in the foot. There was bleeding on cutting the skin to test circulation. The foot was pale but blanched under the nail beds. Paravertebral sympathetic block was performed and the foot became pink and warm.

Postoperatively the patient was given papaverine and the head of his bed was elevated 6 inches. Six hours later pulses were present in arteries of the foot. The wound was healed by the fourteenth postoperative day and on the thirty-fifth day the patient left the hospital with no complaints.

SUMMARY

A traumatic aneurysm is reported due to tumor of the bone.

The surgical treatment is described.

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Sacrococcygeal Chordoma

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BECAUSE sacrococcygeal chordoma continues to be a comparatively rare tumor, and because of its unfavorable prognosis, a case of this type will be reported herein.

Gentel and Coley[1] reported seven sacrococcygeal chordomas of 31,099 cases of cancer at the Memorial Hospital, New York, from 1920 to 1943. In 1933, 168 known cases had been recorded in the literature.[2]

Our case is the only one found in 35,000 admissions to the Pfeiffer Surgical Clinic of the Abington Memorial Hospital from 1937 to 1954, a period of seventeen years.

Chordomas are acknowledged to be derived from notochordal tissue abnormally placed or traumatically released *in situ*. It is a slow-growing tumor but rarely if ever curable. It recurs locally in most cases but does metastasize in about 10 per cent. One of the most striking features of a chordoma is its local destructive effect on bone. The fact that they are seldom diagnosed early warrants some consideration. Differential diagnosis must be made from other tumors in this area, such as dermoids, sarcoma, carcinoma of the rectum, tuberculosis of the bone, female pelvic tumors and abscess. Pain in the region of the sacrum or coccyx, or lumbago may when investigated reveal a rubbery-like mass in the midline either anterior or posterior to the sacrum. Since bone destruction is an early manifestation, x-ray examination is a most important diagnostic aid. The picture is somewhat typical showing destruction and rarefaction and occasionally areas of calcification. Aspiration biopsy should confirm the diagnosis.

CASE REPORT

E. G., a forty-three year old white man, was admitted on June 25, 1952, with the chief complaint of a feeling of fullness in the rectum. For the preceding six months the patient had noticed a bearing-down sensation in the rectum which was more obvious on standing or when riding in a car. Bowel habits showed no significant changes. Occasional loose stools were attributed to drinking beer. The patient had noticed no blood or mucus in his stools; they seemed of normal color. Bowel movements caused some pain, and recently the patient had noticed some difficulty in starting stream when urinating. He attributed the latter to the medicine given him by his family doctor for relief of pain.

In 1941 thoracotomy had been performed for drainage of lung abscess. Since then there had been no symptoms of chest disease. The patient had fractured his ankle four years previously and had had questionable trauma to the end of his spine. No x-rays of the sacrococcygeal region were taken at that time.

Family history was negative.

Physical examination was essentially negative except for the rectum. A tumor mass about the size of a lemon was palpated in the posterior midline anterior to the coccyx. It seemed fixed in the hollow of the sacrum. It was elastic in consistency and non-tender. No blood or mucus was noted on the examining finger and there were no peripheral nerve signs or symptoms. A sigmoidoscope was passed easily above the tumor mass. The mucous membrane appeared normal throughout.

X-ray report on June 27, 1952, revealed the following: Anteroposterior view showed far advanced destruction of the lateral wings of the fourth and fifth sacral segments and part of the third segment. On this view there was a globular shadow in the pelvis which could have been that of a filled bladder. However, follow-up examination performed immediately after the patient voided still showed this globular shadow. It would be quite unusual for an empty bladder to have cast such a shadow. We could not determine whether the bone destruction of the sacrum was due to a lesion of the sacrum itself or to pressure erosion. (Fig. 1.)

Operation was performed on June 30, 1952, five days after admission, this time interval being used for studies and bowel preparation. Excision of the sacral tumor was performed

FIG. 1. X-ray taken before operation showing soft tissue shadow of growth and bone destruction in the sacrum.

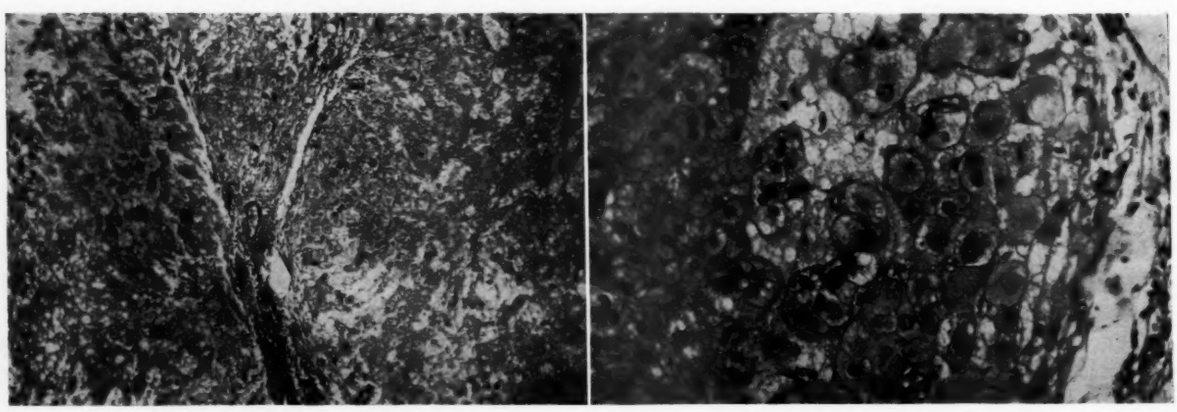

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FIG. 2. Microscopic section (low power) showing masses of tumor cells separated by thin trabeculae of connective tissue.

FIG. 3. Microscopic section (high power) showing many tumor cells floating in mucoid secretion. Nuclei are hyperchromatic and bizarre.

under spinal anesthesia. With the patient in the prone jackknife position, a midline incision was made with the Bovie knife over the sacrum and extending downward to just above the anal sphincter. A lateral cross incision was made between the sphincter and the tip of the coccyx, dividing the coccygeal anal ligament. A tumor about the size of a lemon was palpated in the hollow of the sacrum. The sacrum was divided with a chisel above the growth at the middle of the third sacral segment. The attached lateral muscles and ligament were divided sacrificing the lower sacral nerves. The bone was widely infiltrated and deteriorated by the tumor mass. The anterior surface seemed to be encapsulated and was separated from the rectum by blunt dissection. A finger was placed in the rectum during this maneuver. The entire mass was then removed including the lower portion of the sacrum and coccyx. The tumor had a

grossly gelatinous encephaloid appearance and was very friable.

The coagulating current was applied to the entire end of the bone and soft tissue with the exception of the surface of the rectum. The wound was packed open with iodoform gauze.

Pathological report revealed the following: Microscopically, lobular masses of tumor cells were separated by thin trabeculae of connective tissue. (Fig. 2.) The tumor cells were quite pleomorphic but most were coarsely vacuolated. Many were floating in mucoid secretion. Nuclei were bizarre, many being misshapen and hyperchromatic. (Fig. 3.) In a few foci the tumor cells were better differentiated and formed compact masses.

The cells proliferate in an invasive manner, invade and destroy cancellous bone, and also invade connective tissues.

Diagnosis was chordoma of sacrococcygeal region.

The postoperative course was uneventful. There was no trouble with either the bladder or anal sphincter. Naturally there was considerable slough due to using the cautery. Half of the packing was removed on the fifth postoperative day and the remainder on the seventh postoperative day. The wound was then irrigated with azochloramide solution and a clear healthy granulating wound was present on the sixteenth postoperative day, the day of discharge from the hospital. X-ray therapy was begun on the third postoperative day. In all, 11,784 skin dose roentgens through four ports were given. Chordomas are considered radioresistant, although the favorable response reported in a few of the younger cases made us believe we should use it.[4]

Follow-up examination at the Clinic on November 10, 1952, showed the wound to be well healed. The patient had resumed full activities about two months previously.

The roentgen report was as follows: "Reexamination of the sacrum shows no further progress of the previously described surgical defect. There has been in fact some re-calcification along the margins of the surgical defect. No new areas of involvement are obvious." (Fig. 4.)

FIG. 4. Follow-up roentgenogram taken November 10, 1952, showing surgical defect.

COMMENTS

Because of the recurring and malignant nature of sacrococcygeal chordomas, the prognosis is disheartening. The average length of life in all cases reported is about four years. It recurs locally in most of the cases and metastasizes in 10 per cent to the inguinal lymph nodes, liver, lungs, pericardium, heart and skin. The extent of surgical treatment therefore remains controversial. Primary radical extirpation by both abdominal and posterior approach, as suggested by Mixter and Mixter,[4] would seem to offer the only hope of cure, although repeated local excision in some cases has given better palliative results.

An operative team composed of the surgeon, orthopedist and neurosurgeon might result in a more thorough local resection.[3]

Until an operative technic is developed which envisages complete removal of the tumor-bearing segments of the sacrum and coccyx, the present unsatisfactory results of surgery will probably not be improved materially. Such radical surgery naturally involves difficulty with regard to bladder or rectal control.

To propose such radical and objectionable procedures as permanent colostomy and ureteroenterostomy in the early stages of this disease (when limited extirpation might be successful) may be considered unduly radical by the surgeon and unacceptable to the patient.

Since 90 per cent of the recurrences are local, it was thought that the use of the destructive coagulating current might be of some value. Therefore it was used in our case. We could not

find any report of such treatment in the literature.

When last seen on July 1, 1955, the patient showed no evidence of recurrence either on physical or x-ray examination.

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Jelinek and co-workers in Vienna compared postoperative results in a series of patients treated with vagotomy alone and a series of patients treated with vagotomy plus partial gastrectomy. They found vastly superior results in those patients who had the combined operation (vagotomy plus gastrectomy). The follow-up period, however, was rather short, varying from one to six years. (*Richard A. Leonardo, M.D.*)

Acute Diverticulitis of the Cecum with Perforation

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ACUTE diverticulitis of the cecum is an uncommon disease. No one surgeon has had a large series; most of the reports in the literature deal with an author's personal experience with one to five cases. Up to January, 1952, only 130 cases were recorded. The surgical management, however, is not simple, and the procedure that is carried out may be fairly formidable. In the hands of the inexperienced person the morbidity, if not the mortality rate, may conceivably be great.

In a large number of books on surgery this disease is not even mentioned. Among the most comprehensive articles in the literature were those written by Anderson,[1] and Vaughn and Narsete.[3] Acute appendicitis is the usual clinical diagnosis. At the time of operation carcinoma of the cecum may be suspected due to the induration encountered secondary to the inflammatory process.

The etiology of cecal diverticula has not been definitely established. At the Mayo Clinic, Anderson found only eight cases in 700 of colonic diverticulitis where the lesion was limited to the cecum, illustrating the low incidence of cecal diverticulitis. Autopsy studies show that only 1.5 per cent of large bowel diverticula occur in the right half of the colon.

This disease may occur at any age. The youngest patient recorded was three years old, and the oldest was sixty-nine. It is encountered most frequently from the third to sixth decades. The sex ratio is approximately 1:1. In nearly one-half of the cases fecaliths were found in the diverticula which were usually located on the lateral or medial wall of the cecum.

Treatment is surgical. The pathologic disease found determines the extent of surgery. Procedures which have been carried out in the cases reported are: simple drainage, excision of the diverticulum, inversion, local resection of the cecum and radical resection.

As for complications following surgery, fecal fistula was most frequent. This was followed by wound infection, sinus formation, pneumonia, abdominal distention and peritonitis.

The over-all mortality rate was approximately 5 per cent. However, the true mortality rate is probably higher as this figure represents only those cases reported from the larger medical centers.

CASE REPORT

A. N. was a twenty-eight year old married woman. On admission in August, 1953, she complained of right lower abdominal discomfort of two days' duration. This was associated with occasional epigastric cramps, nausea and backache. There was no history of vomiting. Bowel movements were regular. She had no diarrhea or urinary complaints. The menstrual history revealed no abnormality. Her last period was normal and had occurred three days prior to hospital admission.

On abdominal examination there was tenderness in the right lower quadrant. A palpable mass was noted which was interpreted as being omentum covering an acutely inflamed appendix. Pelvic examination revealed a normal uterus and tenderness in the right vault. Rectal examination was negative. The patient's temperature was 100°F., and the white blood count 17,600. Urinalysis was negative. The preoperative diagnosis was acute appendicitis.

Operation was performed through a right lower paramedian incision. The cecum was indurated and edematous, and a cecal mass about 5 cm. was felt. The appendix, terminal ileum and the remaining large bowel were normal. Clinically it was impossible to rule out carcinoma of the cecum. The lymph nodes in the ileocecal mesentery were enlarged up to 3 cm. The edema extended retrocecally. No frank pus was noted. The cecum was opened and a biopsy was taken for frozen section. The posteromedial wall of the cecum presented an opening 2 cm. in diameter extending retrocecally.

At this point one could not be sure whether he was dealing with a pure inflammatory process or inflammation secondary to a perforating tumor. The pathologist's report of the frozen section was "acute inflammation." Even then one could not be too certain about the diagnosis because a representative section might not have been given to the pathologist; therefore tumor could not be ruled out. In order to remove the gross disease a portion of the ascending colon, cecum and terminal ileum was resected together with the ileocecal mesentery, and open end-to-end anastomosis was performed between the terminal ileum and the remaining ascending colon.

The patient was given penicillin and streptomycin for three days postoperatively and then oral aureomycin. The postoperative course was uneventful except for superficial wound sepsis. She was discharged on the fourteenth postoperative day. The pathologic diagnosis was cecal diverticulitis with perforation, and pericecal abscess.

In retrospect, if drainage alone had been carried out, the postoperative complication might have been a fecal fistula and further surgery would have been necessary. Simple excision was impossible since the inflammation and induration present obscured the diverticulum. Neither drainage nor simple excision would have been adequate if the pathologic condition had been due to a perforating tumor.

COMMENT

The benign postoperative course of this patient was due in no small measure to the broad-spectrum antibiotics which were given. A good anastomosis with postoperative antibiotics greatly minimizes any complications which may ensue following resection on an unprepared bowel.

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An Automatic Self-threading Boomerang Needle Holder

THOMAS KIRWIN, M.D., *New York, New York*

ALTHOUGH the credit for invention of the boomerang surgical needle holder is usually accorded to the late Hugh H. Young of Baltimore, actually the idea was not original with him. Nor did Young—at least so far as I have been able to determine—ever lay specific claim to such credit. As a matter of fact such a needle holder was described and employed as a part of his surgical armamentarium by Albarran, the distinguished French colonial urologist, in the first years of the twentieth century.

Young's model was a considerable advance upon that of Albarran. After it had been in the hands of urologic surgeons (both here and abroad) for a comparatively short time, a number of those who had used it thought out improvements in the design of the working parts which instrument makers produced from time to time, often applying the name of the "improver," either bracketed with that of Young or, as time went on, dropping Young's name altogether in favor of some more recent modifier of the original design.

Thus the modification which the Australian, S. Harry Harris, displayed when a guest of the American College of Surgeons at Chicago in 1929, soon came to be known as the Young-Harris boomerang needle holder. When Terrance Millin described it in his *Retropubic Urinary Surgery*, published in 1947, he mentioned that "the handle is of the standard pattern originally introduced by Young" and "the ligature carrier is of a pattern devised by Harris." Despite Millin's statement most recent British writers speak of "Millin's boomerang needle" and describe various improvements but always along the general lines of Young's design.

The present writer during many years experience in urologic surgery has had occasion to employ boomerang needles of the Young pattern, but always found them not entirely satisfactory. After analyzing the factors which led to this dissatisfaction, the problem was taken to the American Cystoscope Makers, Inc. The result of this collaboration is shown in the accompanying drawings in which Mr. William P. Didusch has illustrated an entirely new type of needle-holder, giving the operator complete control of a handle which is an integral part of the instrument, with automatic "feeding" of the suture material.

The drawings are so detailed yet so clearly presented that no elaborate explanation of the function of the different parts is necessary. (Figs. 1 to 5.)

DESCRIPTION OF INSTRUMENT

The component parts of the new boomerang needle are illustrated in Figure 1. A bobbin holding suture material is enclosed in a metal case. The needle (on the distal end of the instrument) has a slot and a forked shaft which carries the suture material to a point where it can be engaged by the slot in the needle. Simultaneous excursions of the needle, with its forked shaft conveying the suture material, can be produced by pressure on the trigger near the instrument's handle. Rotation of needle and forked shaft is made possible by a disc which allows it to be placed in any desired position without changing the angle of the handle. The effect of such pressure on the trigger-excursions of needle and forked shaft is illustrated in Figure 1B.

In Figure 1C the engagement of the suture material in the needle slot is shown, and in Figure 1D we see how release of the pressure on the trigger permits the needle to draw the suture material from the forked shaft.

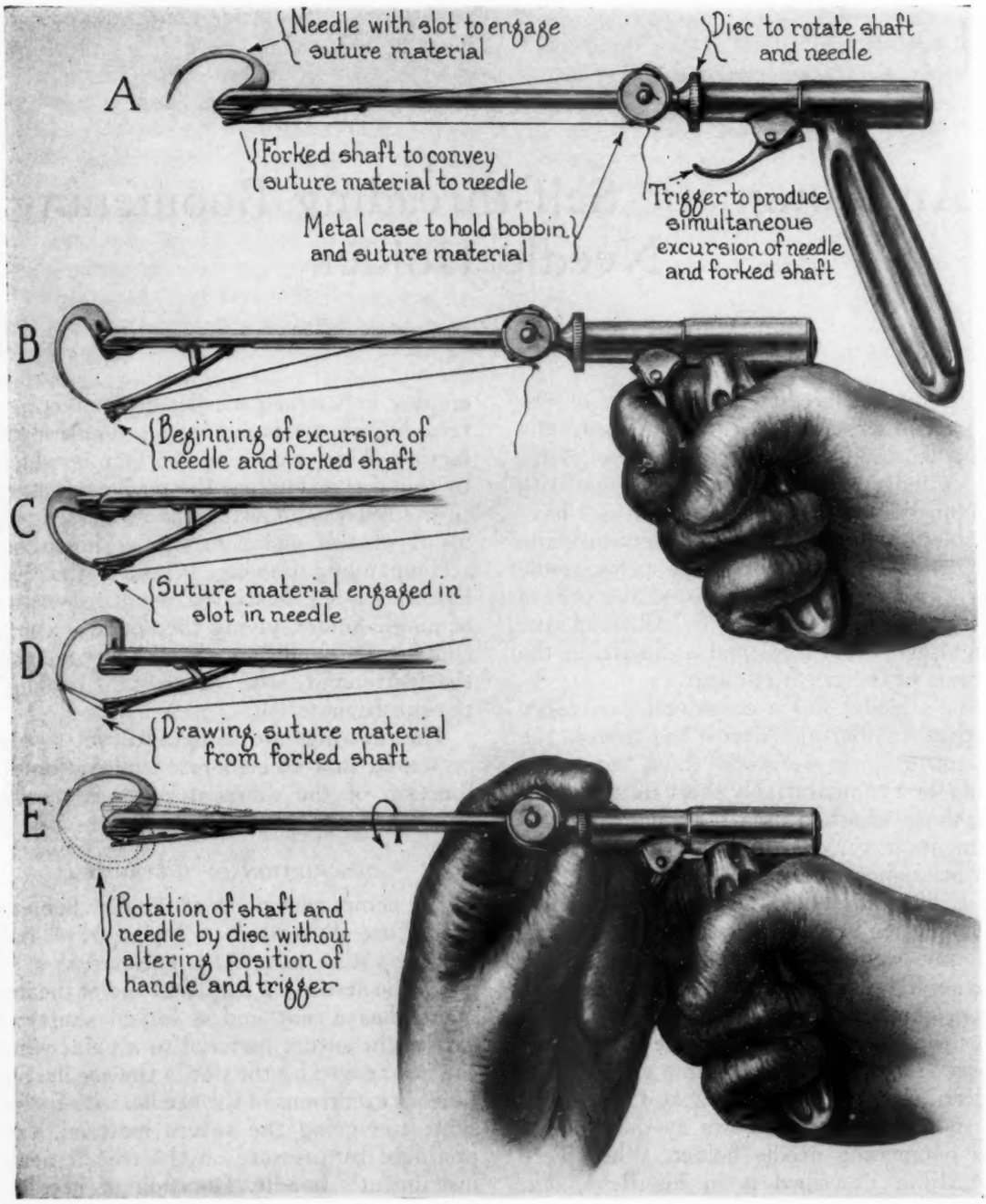
Needle with slot to engage suture material
Disc to rotate shaft and needle
A
Forked shaft to convey suture material to needle
Trigger to produce simultaneous excursion of needle and forked shaft
Metal case to hold bobbin and suture material
B
Beginning of excursion of needle and forked shaft
C
Suture material engaged in slot in needle
D
Drawing suture material from forked shaft
E
Rotation of shaft and needle by disc without altering position of handle and trigger

FIG. 1. A, automatic boomerang needle holder with component parts: metal case contains bobbin with suture material. On the distal end of instrument is a needle with a slot and forked shaft which carries the suture material to a position where it can be engaged by the slot in the needle. A trigger near the instrument's handle produces simultaneous excursions of the needle and the forked shaft holding the suture material. A disc makes possible the rotation of needle and forked shaft to any desired position without changing the position of the handle. B, shows how pressure on the trigger produces excursion of the needle and forked shaft. C, shows suture material engaged in slot on needle. D, as pressure on trigger is released, needle draws suture material from forked shaft. E, needle and forked shaft are rotated by means of disc, without altering position of handle and trigger.

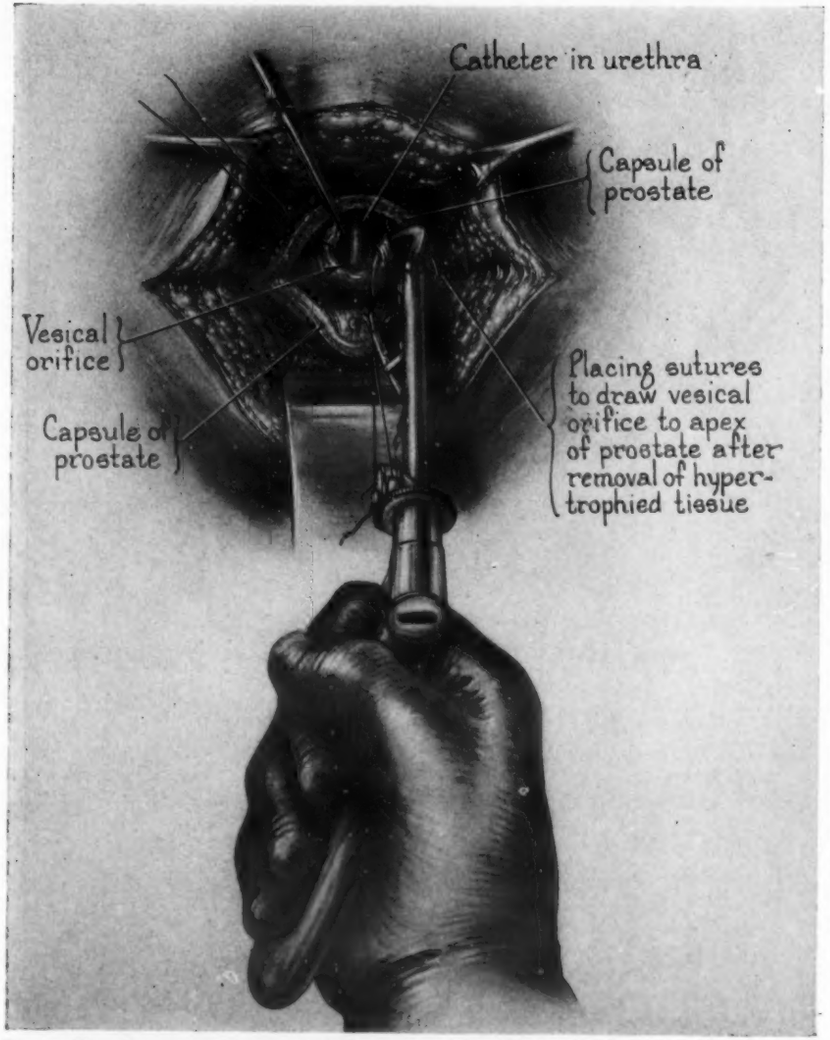
Catheter in urethra
Capsule of prostate
Vesical orifice
Capsule of prostate
Placing sutures to draw vesical orifice to apex of prostate after removal of hypertrophied tissue

FIG. 2. Employment of automatic boomerang needle holder in perineal prostatectomy. Sutures are being placed to draw vesical orifice down to apex of prostate after removal of hypertrophied tissue.

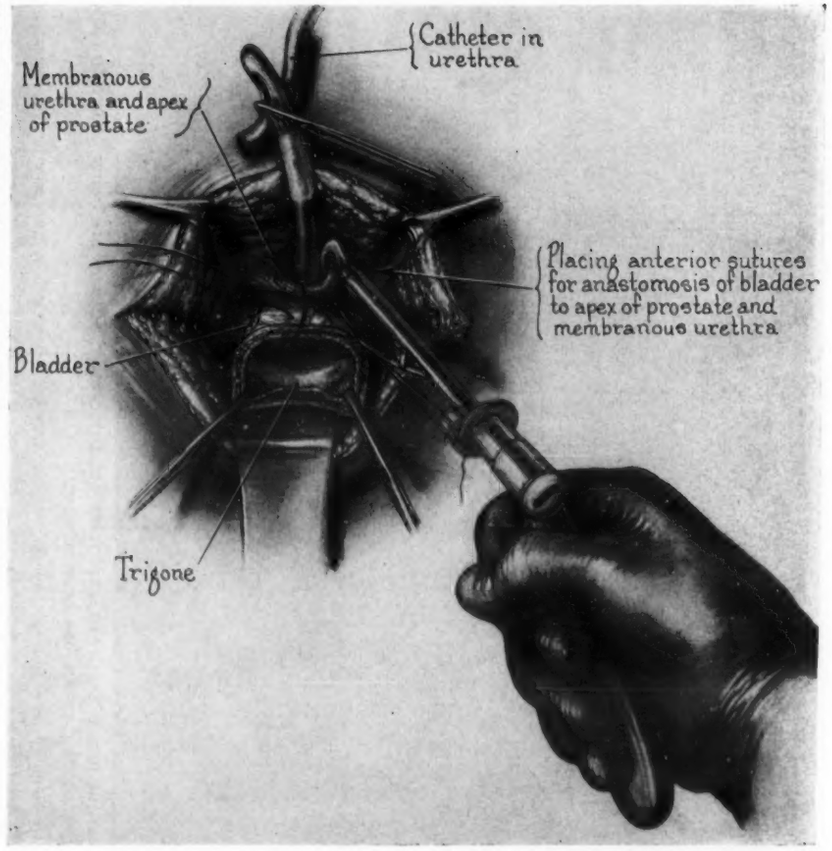
Membranous urethra and apex of prostate
Catheter in urethra
Placing anterior sutures for anastomosis of bladder to apex of prostate and membranous urethra
Bladder
Trigone

FIG. 3. Employment of automatic boomerang needle holder in radical operation for prostatic carcinoma. Sutures are being placed to anastomose the bladder to apex of prostate and to membraneous urethra.

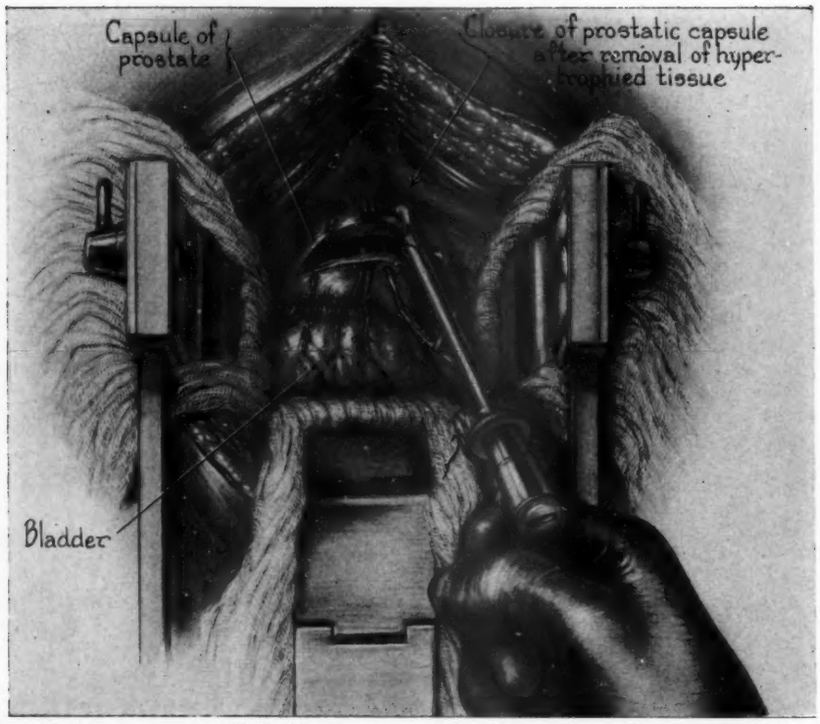

FIG. 4. Use of automatic boomerang needle holder in retropubic prostatectomy. Sutures are being placed to close the prostatic capsule after the hypertrophied tissue has been removed.

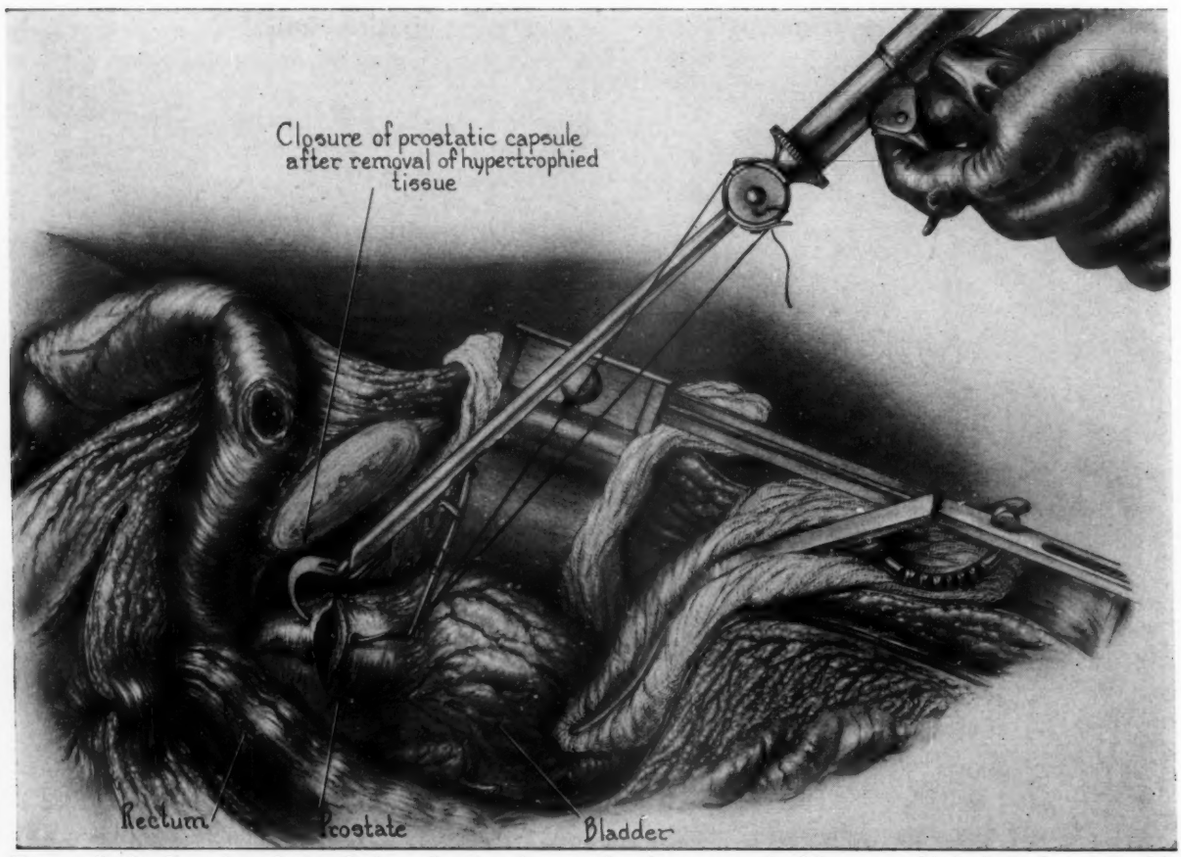

FIG. 5. Sagittal section shows closing of prostatic capsule after removal of hypertrophied tissue in retropubic prostatectomy.

Needle and forked shaft can be rotated with the disc without altering the position of handle or trigger, as is shown in Figure 1E.

The automatic self-feeding feature of this instrument has many advantages. It assures positive threading of the suture material to the needle; it permits the operator to control the excursion of the needle without the help of his assistants and the operative field is not obscured. Lastly, it saves time.

The bobbins for this instrument with suture material of plain and chromic catgut in sizes No. 0000, No. 000, No. 00, No. 0 and No. 1, packaged sterile in tubes.*

Although this instrument† is adapted for use in a wide variety of surgical operations upon deep body cavities and other hitherto inaccessible operative fields, the author being a urologic surgeon shows it here used in interventions upon the prostate. However, its availability in other procedures will be readily recognized by any surgeon.

* Supplied by Davis & Geck, Inc., Danbury, Connecticut.

† Obtainable from the American Cystoscope Makers, Inc., New York, N. Y.

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The average case of Hirschsprung's disease will be cured by one-stage excision of that part of the colon which showed no peristaltic activity on x-ray, together with removal of the "narrowed portion" of the rectosigmoid, followed by restoration of intestinal continuity. It is wise to save the rectum because thereby the nervi erigentes are protected. (*Richard A. Leonardo, M.D.*)

A New Plastic Needle

SIDNEY HIRSCH, M.D., *Cedarburst, New York*

THE increased use of plastic tubing for intravenous therapy has inspired the search for a simpler method than that previously employed for the insertion of this tubing. The previous method of inserting the tubing through the needle that was to enter the vein necessitated the use of an unusually large gauge needle, either a No. 15 or No. 16 gauge, and then the cumbersome method of withdrawing the needle and adapting the plastic tubing by one means or another to the intravenous medication.

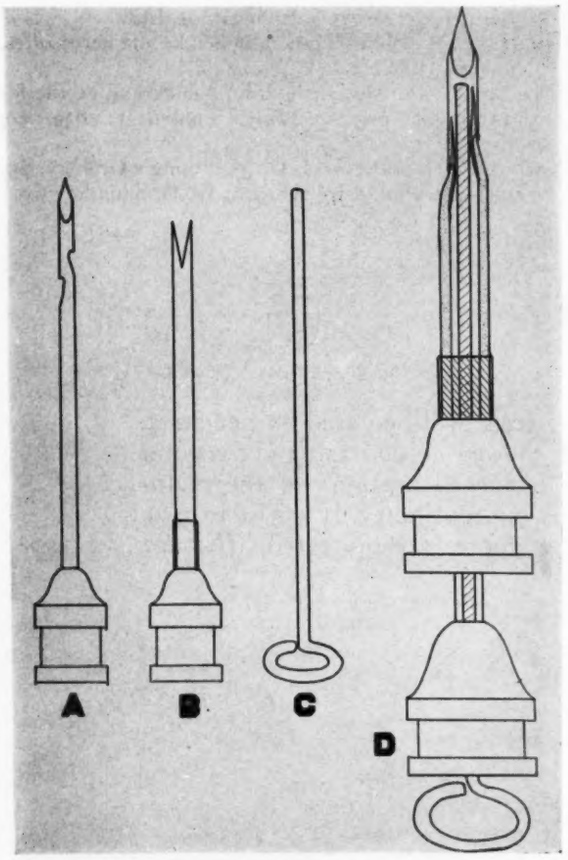


FIG. 1. Diagrammatic sketch of needle. A, slotted needle No. 19 gauge; B, plastic tubing showing tapered ends and luer hub attachment; C, stylet for No. 19 gauge needle; D, assembled needle.

The needle described herein simplifies the technic considerably. (Figs. 1 and 2.) It eliminates the need for a "cut-down." By fitting the plastic tubing into ingeniously precut slots in a No. 19 gauge needle, no shoulder of the plastic tubing remains outside the needle, thus permitting the tubing to follow the needle easily through the skin into the vein. Furthermore the needle stylet holds firmly the cut ends of the plastic tubing which are within the slotted holes of the needle. After insertion of the whole unit into the vein, the stylet is withdrawn, the needle pushed about ¼ inch further into the vein while holding the tubing and its hub firmly; the needle is turned about 90 de-

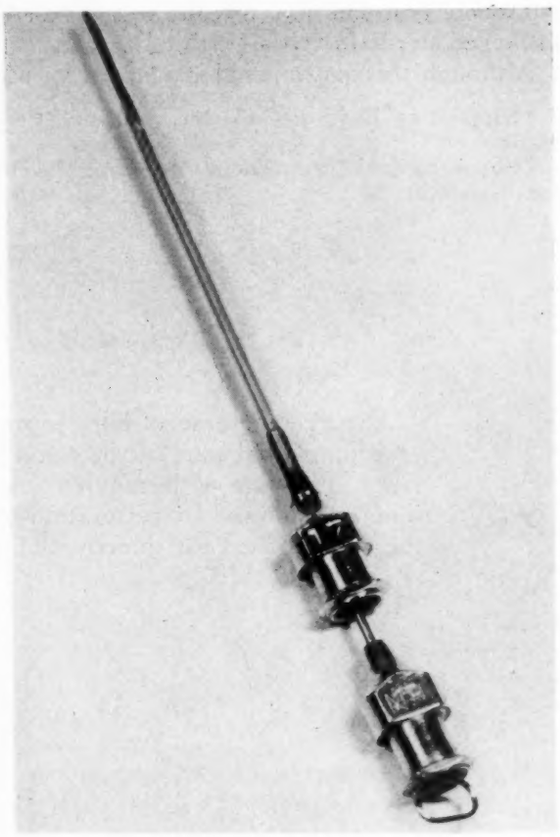


FIG. 2. Assembled needle.

grees either to the right or to the left and withdrawn.

A luer hub is already part of the plastic tubing so that the adapter commonly part of any intravenous or transfusion set can be attached directly to this hub and the hub fixed to the skin with adhesive.

The plastic tubing can remain in the vein for days without causing any venous or skin irritation. When desired, it is withdrawn from the vein without any difficulty.

Acknowledgment: I wish to thank Mr. George Utley, designer, for his assistance in developing this needle.

If the portion of colon containing cancer is ligated several inches above and below the growth prior to its mobilization for purposes of excision, recurrences are less apt to occur. The bowel lumen and the vessels and lymphatics in its wall usually contain cancer cells. If the loop is isolated by ligatures first, one is less apt to disseminate the disease and end-to-end closure of colon should not be followed by recurrence at the anastomotic site. Also, the local arteries and veins supplying the loop should be ligated before excision to prevent dislodging of the cancer cells during mobilization of the loop. These two changes in the operative technic will probably reduce the incidence of postoperative recurrence in segmental resection of the colon for early malignancy. (*Richard A. Leonardo, M.D.*)

BOOK REVIEWS

Atlas of Operative Technic, Anus, Rectum and Colon. By Harry E. Bacon, M.D. and Stuart T. Ross, M.D. 301 pages, 403 illustrations. St. Louis, 1954. C. V. Mosby Company. *Price* $13.50.

The authors tell the reader that although many textbooks are available today, the rapid pace of modern surgical practice leaves little opportunity to the surgeon for extended study. To fill this gap the work on the Atlas was begun some seven years ago. It is a pictorial presentation illustrating the consecutive steps in the various operations in this field, accompanied by short step-by-step descriptions. After General Considerations (Surgical Anatomy, Pre- and Postoperative Care in Anorectal Surgery and in Colonic Surgery, Anesthesia, Basic Instrument Set-Up, etc.) the work proper begins with text on the left hand page and illustrations on the right hand page. The drawings are well done and the steps in surgical technic are easy to follow and comprehend.

This book is recommended to all surgeons—from intern to attending—who are interested in surgery of the anal canal, rectum and colon.

Urology. Edited by Meredith Campbell, M.D. with the collaboration of 51 contributing authorities. Three volumes, 2,356 pages, 1,148 figures. Philadelphia, 1954. W. B. Saunders Company. *Price* $60.00 per set.

This review could end with the statement that, in the opinion of the writer, this three-volume work edited by Meredith Campbell with the collaboration of fifty-one contributing authorities, is the most complete and up-to-date work on urology at the present time.

Space does not permit us to consider the work of each author. Every collaborator is outstanding in his specialty. Each contribution is far above the average; many of the chapters are scientific classics. We searched for defects and found none. It has everything a reader or student can desire. The subject is thoroughly covered in the most minute detail. The illustrations are above the average. There is no padding.

The publishers are to be congratulated for bringing out this typographically beautiful volume, and Dr. Campbell commended for his skillful editing.

These three volumes are well worth their cost. This is a work that should enjoy a substantial sale and prove an endless scientific urologic joy to the reader.

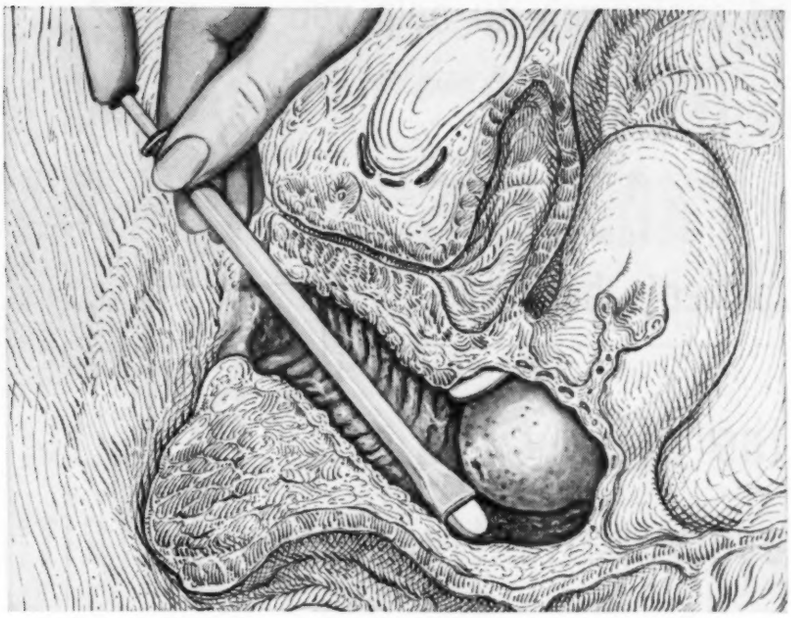
Textbook of Operative Gynecology. By Wilfred Shaw, M.D. 444 pages, 382 figures, some in color. Edinburgh and London, 1954. E. & S. Livingstone, Ltd. *Price* $19.00.

Wilfred Shaw had just reached the top rung in the ladder of his surgical specialty when he was mortally striken with a disease, the outcome of which he knew to be inevitable. He filled his remaining days with the completion of this textbook of operative gynecology. Apart from a few extra illustrations, we are told nothing has been altered or added.

The author was widely known as a skillful operator and recognized as one of the outstanding British gynecologists of his time. He has written an exceptional work. The reviewer cannot decide whether he likes the text or the well done illustrations most. Both show the hand of the professional.

We believe that the young surgeon learns the technical part of his calling at the operating table, starting as a suture and instrument nurse and gradually advancing until he is competent to undertake the various operations himself. But, in addition to this training, it is a must that he know the anatomy involved, the pitfalls to avoid and the various technics that have proved by time and trial to be the most successful.

This beautiful book is a volume that every young surgeon who aspires to become a recognized gynecologist should own and study, and then restudy, both text and illustrations. No matter how long one has been practicing the specialty, this book will be of value.



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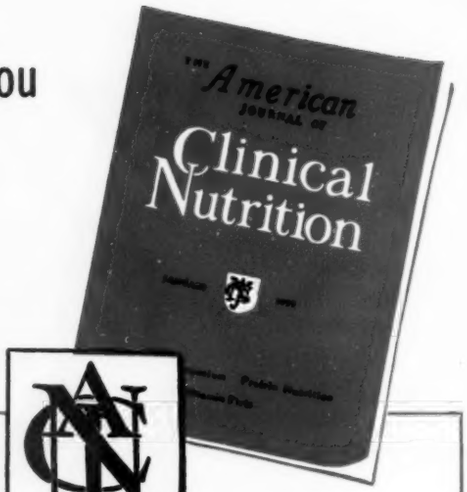

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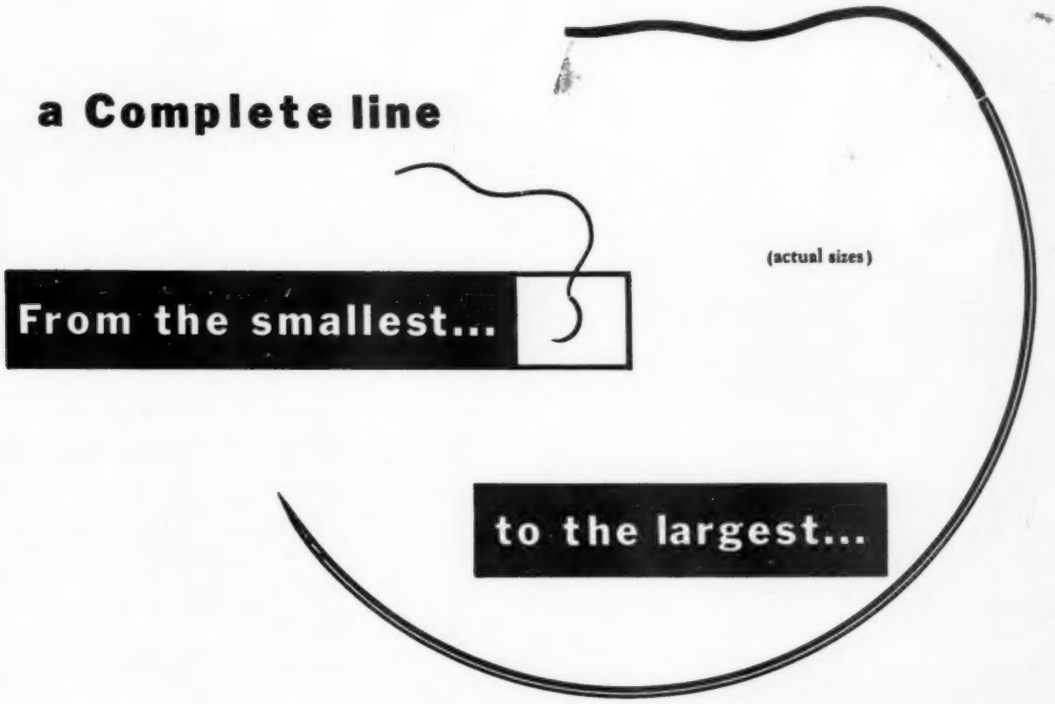

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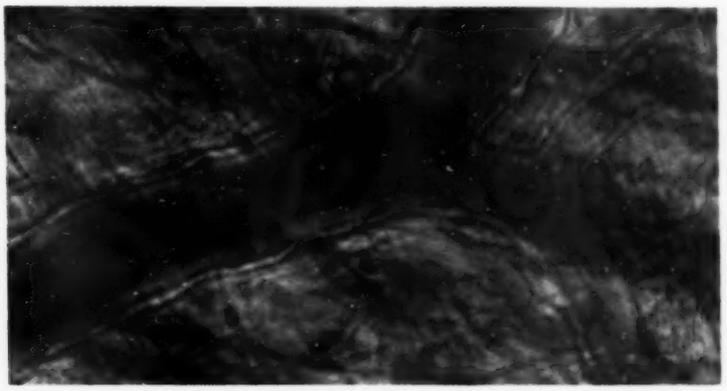

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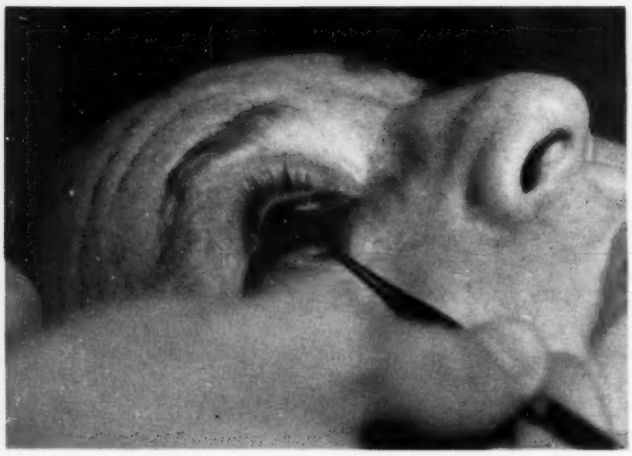

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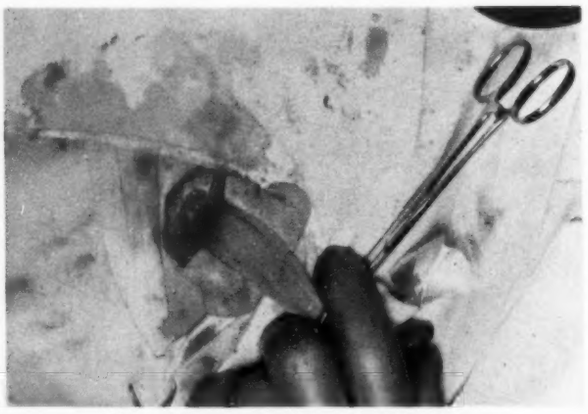

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  1 to 5 mg. orally t.i.d.; if bleeding ensues, dosage may be increased 1 to 5 mg. orally every three or four hours; if bleeding persists, oral dosage should be supplemented with one ampul (5 mg.) daily.
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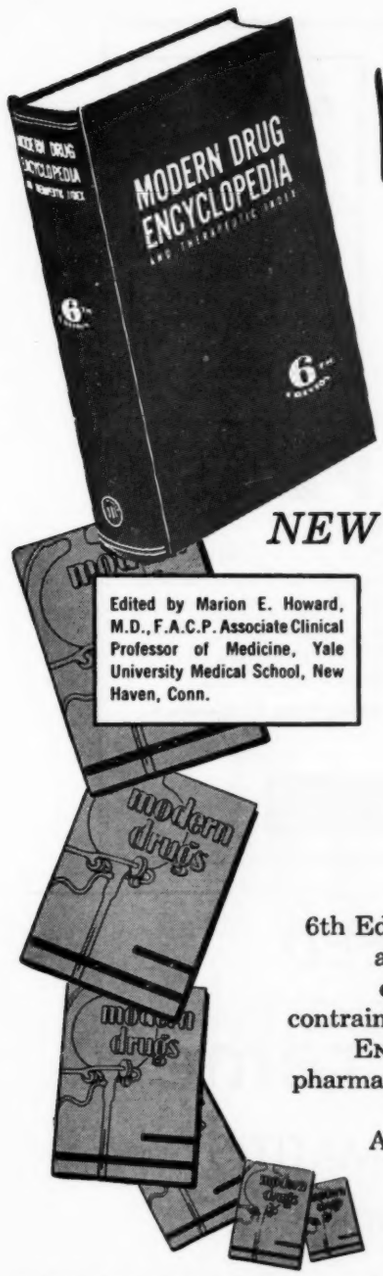

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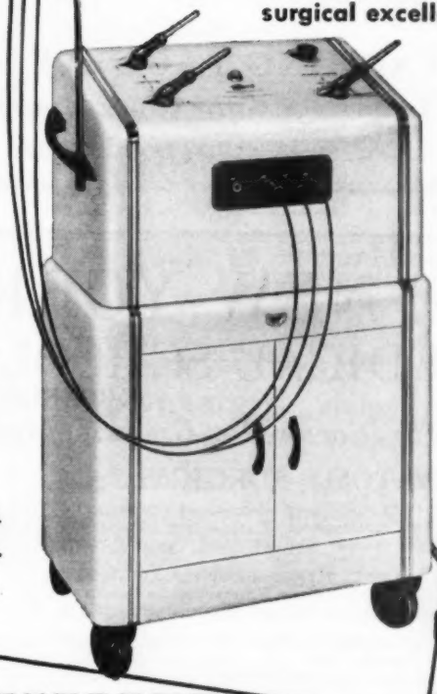

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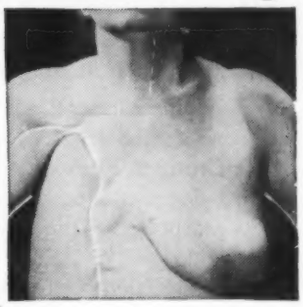

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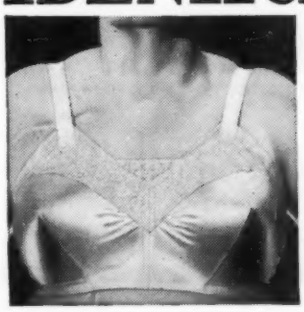

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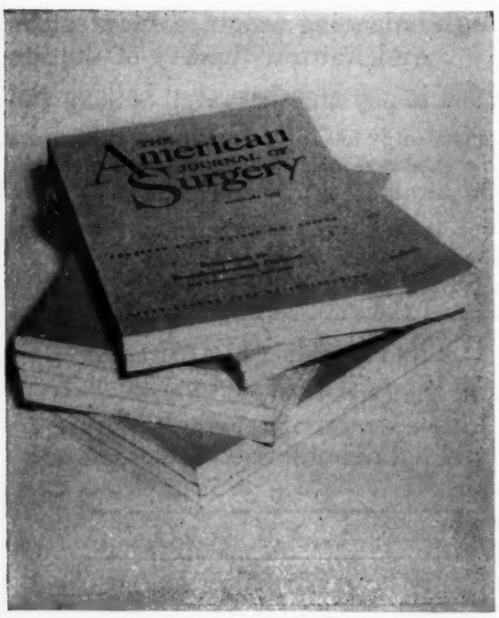

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The Argentine chapter of The International College of Surgeons will hold the Second Panamerican Congress of the International College of Surgeons simultaneously with the Eighth Argentine Meeting of Thoracic Surgery on October 22 to 26, 1955, at Mendoza, Argentina. For further information communicate with the Secretary, Caseros Av. 2153, Buenos Aires, Argentina.

A TEN-POINT code of ethics governing the preparation of medical advertising and sales promotional material was adopted unanimously by the Board of Directors of the Pharmaceutical Advertising Club of New York and the Midwest Pharmaceutical Advertising Club at a joint meeting held in Atlantic City during the annual meeting of the American Medical Association.

The code, presented by G. L. Long, Advertising Manager of Mead Johnson & Company, recognizes that ethics in pharmaceutical advertising are based upon conscience, faith, trust and integrity.

The code of ethics, adopted by the Boards of Directors of the clubs, encompasses the following points: (1) Pharmaceutical advertising should not, by intent or inference, defraud, deceive or mislead the reader. Any statement of fact should be accurate and supportable by adequate and acceptable scientific evidence. Any statement of rationalization or speculation should not be presented as a fact—stated or implied. (2) Any statement made in pharmaceutical advertising should be believable and provable. Adequate and objective evidence should be unquestionable. Advertising should not exceed claims which are supportable by such evidence. (3) A pharmaceutical advertising program should give a proper perspective of a product's role in patient management, such as, properties, indications, limitation of use, advantages and disadvantages. (4) Quotations or excerpts, abstracts or paraphrasing from published medical papers or texts, personal communications, scientific exhibits or speeches shall not by themselves change or distort the true meaning intended by the authors. Such material shall not be used if it is not consistent with later known findings, observations or conclusions of the authors or investigators. Such material shall not be cited as representing general medical opinion if it reflects a point of view held only by an individual worker or group. (5) Comparisons with competitive pharmaceutical specialties, aimed at establishing product superiority, are fair and proper if: such comparisons are supported by acceptable evidence; such comparisons are constructive and on a sound medical basis; such comparisons do not refer disparagingly to the safety of the competitive product. (6) A trademark is private property, and it can be used legally only with the consent of the owner of the mark. (7) The medical profession should be given information on the clinical use of a new drug, or a new use for an established drug, before the release of such information to the lay public. (8) Prescription drugs should not be advertised to the lay public. (9) Statements by a physician should not be used in advertising to the lay public. (10) Physicians, or actors purporting to be physicians, may appear in institutional advertising to the lay public, but not in product advertising to the lay public.

Will rheumatic fever and rheumatic heart disease become the next major scourge of childhood to yield to the advances of medical science? The apparent success of scientific research in developing a vaccine against poliomyelitis makes this a question of immediate import to all Americans.

True, the problems of rheumatic fever prevention differ in many respects from those of polio prevention. We cannot go about preventing rheumatic fever directly now, because the precise cause of the disease is not yet known, as in polio;

CONTINUED ON PAGE 50

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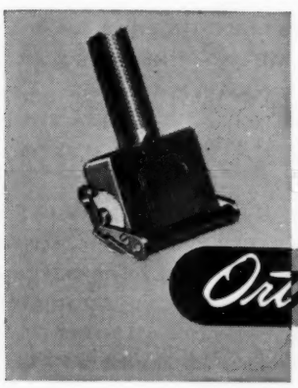

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one attack, moreover, does not confer immunity, as in polio, but rather makes the victim more susceptible to repeat attacks. Scientists supported by the AHA research program and by other groups are now actively seeking the answers to these and many other unsolved problems of rheumatic fever.

Despite these difficulties, however, the medical knowledge is at hand today to make rheumatic fever a rare disease. It has been confirmed repeatedly that prompt and adequate treatment of streptococcal infections can prevent most first attacks of rheumatic fever—that continuous prophylaxis against streptococcal infection can protect most people who have had rheumatic fever against the repeat attacks which are so often responsible for initiating or intensifying heart damage. In penicillin, other antibiotics and sulfa drugs, the medical profession already has weapons which can rout most if not all new outbreaks of rheumatic fever.

THE National Fund for Medical Education has produced its first documentary film, "Danger at the Source," it was announced by the Fund's president, S. Sloan Colt of New York.

Prepared as a public service by Fox Movietone, the thirteen and a half minute film tells the story of medical education in America and is narrated by Ray Middleton.

"Filmed in medical schools and teaching hospitals, supervised by leading medical educators, 'Danger at the Source' highlights the glories and the hard work of the American brand of medical teaching that has made ours the healthiest nation in the world," Colt said.

"Our first film will be a potent aid in bringing home to the American public the critical importance of the nation's 81 medical schools, the seed-bed of all medical care. These schools face the threat of diminishing funds to carry on their teaching programs. Medical education and the need to support it as a free and progressive force for the national welfare is the business of all of us. I hope the film will make this obligation apparent to all who view it."

The National Fund for Medical Education was founded in 1949 to gain private support for the medical schools. The Fund has already distributed nearly seven million dollars in unrestricted grants to the eighty-one schools. The need—and the goal—is ten million dollars each year.

The National Science Foundation has announced 297 grants totaling approximately $2,810,000 awarded during the quarter ending March 31, 1955, for the support of basic research in the natural sciences, for conferences and studies on science, scientific information exchange, for the scientific manpower register and for attendance at scientific meetings.

This is the third group of awards to be made during the fiscal year 1955 by the Foundation for the support of basic research and related matters. Since the beginning of the program in 1951, over 1,500 such awards have been made, totaling about $15,560,000.

APPROXIMATELY 38,000,000 Americans are regular cigarette smokers, although a million and a half have quit smoking entirely in the last eighteen months, according to estimates based on a representative sample of about 40,000 persons surveyed by the U. S. Bureau of the Census for the National Cancer Institute of the Public Health Service, Department of Health, Education and Welfare.

The data will be of value to National Cancer Institute statisticians and physicians who are studying the incidence of cancer in the population, particularly lung cancer, in relationship to smoking habits. A number of laboratory and field studies are being done by the Public Health Service, and by other agencies and institutions with the aid of Federal grants or other financial support. These studies are designed to provide more information on the question of whether or not lung cancer is related to cigarette smoking.

A report containing more detailed findings of the Census Bureau survey is in preparation but will not be ready for several months.

The figures released jointly by the Census Bureau and the National Cancer Institute are preliminary results based on interviews conducted early this year by census takers with both men and women over seventeen years of age in the urban non-institutional population. The survey covered 230 areas consisting of about 450 counties scattered through all regions of the country.

CONTINUED ON PAGE 52

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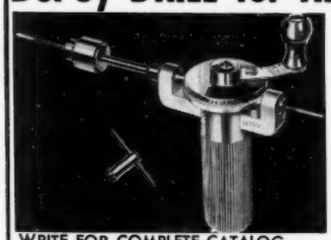

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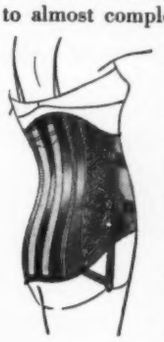

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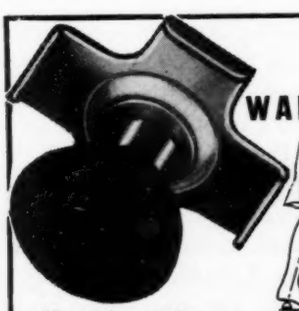

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NOTES AND QUOTES

Not all advertising has become more spectacular with the passing years. In fact, the medical advertisements of today seem pallid compared with those produced half a century ago.

The January 1906 issue of The Yale Medical Journal *carried an advertisement for an extract of red bone marrow. This extract, according to the advertisement, "has in it the elements that are needed by those afflicted with anemia, chlorosis, marasmus, rachitis, malaria and other diseases that the physicians meet with daily." Another advertisement in a 1905 issue of the journal said that "physicians prescribe" a pure rye whiskey sold in "square bottles only."*

That same year The Yale Medical Journal *had an advertisement for a hypophosphate syrup which, it said, was beneficial "in the treatment of Anemia, Neurasthenia, Bronchitis, Influenza, Pulmonary Tuberculosis, and Wasting Diseases of Childhood and During Convalescence from Exhausting Diseases."*

A display of these advertisements is currently featured in an exhibit at the library of the Yale University School of Medicine and shows the history of the journal and its successor, The Yale Journal of Biology and Medicine.

The history of the fight against infantile paralysis can be traced in the issues of these two journals. The lead article of the first issue of The Yale Medical Journal, *dated November 1894, is one of the earliest descriptions of a polio epidemic. Written by Dr. C. S. Caverly, of Rutland, Vermont, it presents the opinion that the disease described in the article is poliomyelitis and that it would eventually prove to be of infectious origin, although not contagious, since only one member of a family was ever attacked by it. An article by Dr. John R. Paul, now professor of preventive medicine at Yale and one of the leading polio researchers in this country, appearing in the July 1938 issue of* The Yale Journal of Biology and Medicine, *describes a group of studies on the epidemiology of this disease in the United States.*

The history of the less successful fight against diabetes can also be traced in the exhibit. An article by Dr. John S. Ely, professor of medical theory and practice at Yale from 1897 to 1906, in the October 1900 issue of The Yale Medical Journal *discusses diabetic coma. He describes himself as a proponent of the acid intoxication theory of diabetic coma and speculated that the acid condition of the blood in such cases is responsible for the peculiar breathing pattern of patients thus afflicted. An article in March 1930 reflects the controversy raging in that period over whether diabetes was caused by an impairment of the ability to oxidize carbohydrates or by an unduly large production of glucose from fat.*

THE American Heart Association and its affiliates have jointly awarded grants-in-aid totalling $723,972.45 to 149 scientists engaged in research in the field of heart and blood vessel diseases, it was announced by Dr. E. Cowles Andrus, president of the Association. The new grants round out the seventh year of the Association's national research support program and are in addition to 110 fellowship awards made known earlier this year. This brings the total allocated by the American Heart Association to underwrite scientific research during the year, beginning July 1, 1955, to $1,418,972. The Association and its affiliates have allocated close to $11,000,000 for research support since the Association became a voluntary health agency in 1948.

The New York Academy of Medicine announces the Twenty-eighth Annual Graduate Fortnight, on the Problems of Aging, from October 10 to 21, 1955. There will be six morning panel meetings, afternoon hospital clinics, twenty evening lectures, four evening panel meetings and a scientific exhibit. Evening lectures and panel meetings will start at 8:30 P.M. All inquiries regarding fees and requests for registration should be addressed to: Secretary, Graduate Fortnight, N. Y. Academy of Medicine, 2 East 103rd St., N. Y. 29, N. Y.

THE American Orthopedic Association elected Dr. David M. Bosworth of New York to serve as its president in 1956–57 at its convention here today. He is director of orthopedic surgery at St. Luke's and Polyclinic Hospitals in Manhattan and at Seaview and St. Vincent's Hospitals in Staten Island, New York.

Dr. Mather Cleveland, chief surgeon of the House of St. Giles the Cripple in Brooklyn, was chosen as treasurer for the same term. Dr. Paul C. Colonna of Philadelphia was installed as president for the coming year.

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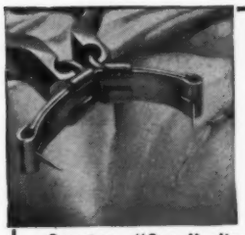

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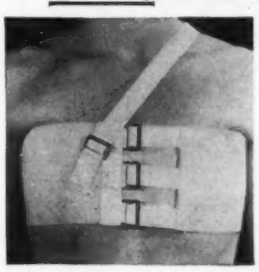

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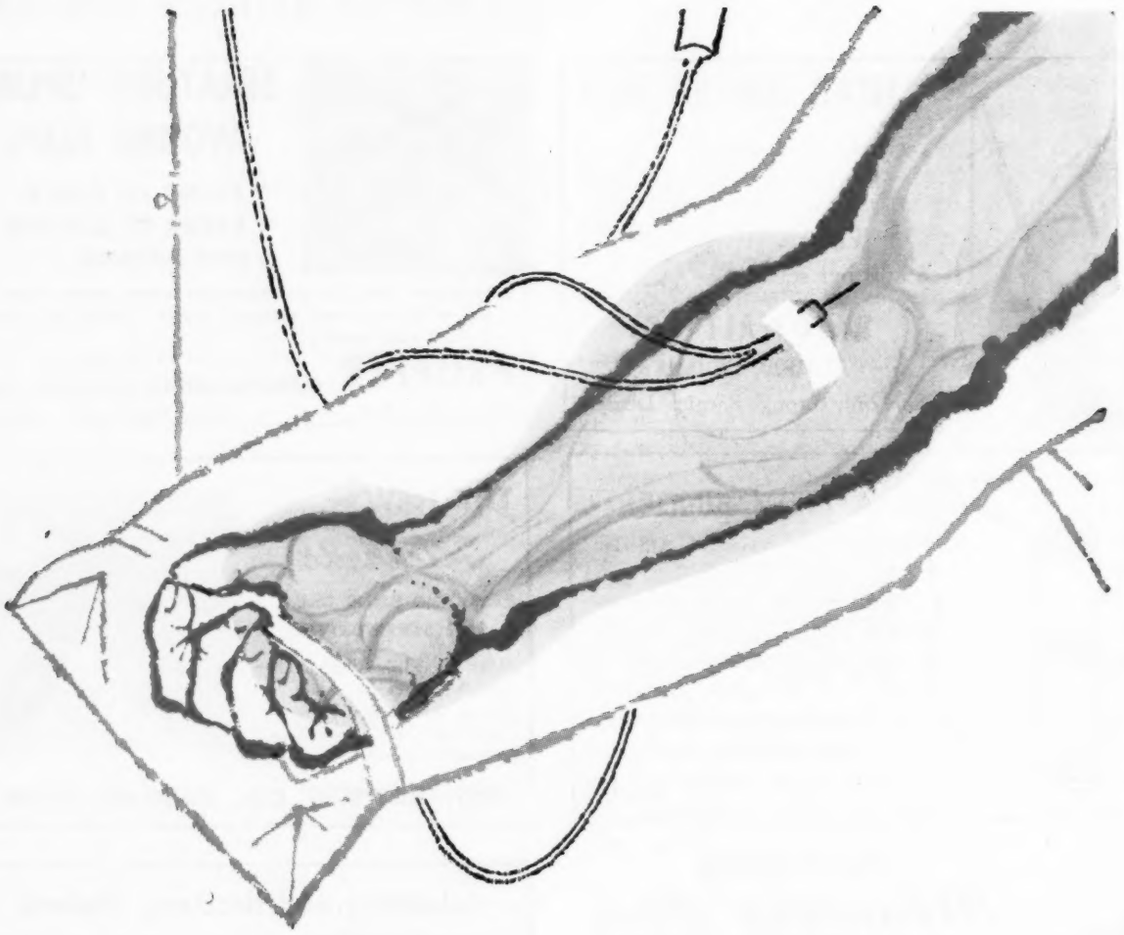

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